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MANUAL OF MEDICINE

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RAYNAUD'S DISEASE (Local Syncope, Local Asphyxia, Symmetrical Gangrene): Its History, Causes, Symptoms, Morbid Relations, Pathology, and Treatment. 5s. net.

A HISTORY OF THE CHRONIC DEGENERATIVE DISEASES OF THE CENTRAL NERVOUS SYSTEM. 6s. 6d.

MANUAL OF MEDICINE

BY

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PREFACE

Advantage has been taken of the demand for a new edition of this Manual to subject the book to a thorough revision from beginning to end, so as to bring it into line anew with the most recent sound teaching. A good deal of additional matter has been introduced, with the result that the new edition is somewhat larger than its predecessor. This is due partly to advances in medical science, which have necessitated fresh articles on such subjects as tropical splenomegaly, trypanosomiasis, and piroplasmosis, and partly to the desire to make the book a still more complete practical guide to the junior practitioner as well as to the student. Two additional illustrations have been introduced, and the figure showing the cortical centres on the lateral aspect of the hemisphere has been redrawn, so as to represent the most recently accepted teaching.

I am indebted to my colleague, Professor Alex. Macphail, for a considerable number of the illustrations, including all those in colour; to Dr. John W. Findlay for the first reading and part of the second reading of the proofs; and to Dr. Findlay and other kind friends for invaluable assistance with the index.

T. K. MONRO.

12, Somerset Place, Glasgow,
May, 1906.

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ERRATUM

Page 133, line 23: *for* ' five or six ' *read* ' two and a half,' as on p. 105.

MANUAL OF MEDICINE

SECTION I

SPECIFIC INFECTIOUS DISEASES

FEVER

The temperature of the body is normally about 98·4° or 98·6° F. (37° C.), but may vary in either direction to the extent of half a degree or more under perfectly healthy conditions. On the whole, the temperature tends to be a fraction of a degree higher in the later part of the day than in the morning.

A temperature persistently below 98° is *subnormal*. This is often seen in patients suffering from chronic ailments and in those recovering from acute disease so long as they remain in bed ; it is then quite a favourable feature. It is the rule in chronic Bright's disease, apart from inflammatory complications or convulsions, and the outlook becomes very ominous in this disease when increasing drowsiness is combined with a tendency to fall on the part of a temperature already subnormal.

When the temperature is abnormally high, it is said to be *febrile ;* if just on the border line between normal and febrile, it is sometimes described as *subfebrile*. There are many varieties of febrile temperatures, and some of them are sufficiently characteristic to indicate the disease—*e.g.*, relapsing fever, tertian and quartan ague.

When the temperature exceeds 106° F., the condition is spoken of as *hyperpyrexia ;* it is very dangerous to life, and

requires prompt treatment, as the high temperature of itself tends quickly to destroy the vitality of the nerve centres. Hyperpyrexia is an occasional complication of rheumatism and some other febrile diseases, and may then occur comparatively early in their course. It is a more familiar occurrence towards the fatal close of some specific fevers and of some cerebral diseases. Its phenomena are very striking when observed as a complication of acute rheumatism, and include stupor or coma, jerking of the tendons (subsultus tendinum), passage of the evacuations into the bed, and great rapidity of the pulse.

Fever and *pyrexia* are regarded by some as synonymous terms. But sometimes the expression ' fever ' is applied to the group of phenomena of which pyrexia or elevation of temperature is the most constant, but which also includes acceleration of the pulse and of respiration, pain in the head and in the muscles, anorexia, a foul tongue, shivering followed by a sense of heat, malaise, and a scanty secretion of concentrated urine. Some of these phenomena—*e.g.*, the rapid pulse—are accounted for by the elevation of temperature, whilst others—*e.g.*, the various pains—are probably due to the poison in the blood which causes the high temperature.

In all ordinary circumstances, when the clinical thermometer is employed, the object is to ascertain as nearly as possible the temperature of the blood. This, of course, has to be done through the walls of the vessels and through the epithelial covering of skin or mucous membrane. The axilla is, on the whole, the best place in adults, and the groin is very suitable in children, care being taken that the skin of the upper arm and chest, or of the thigh and abdomen, are accurately in contact around the bulb of the thermometer. In general practice the observation is often taken in the mouth, but, apart from sentimental objections, there is the risk of fallacy from the mouth having previously been kept open, or from the recent taking of hot or cold food or drink. The rectum gives very accurate results, and the reading is about half a degree Fahrenheit higher than the corresponding figure in the axilla. As a rule, it is

sufficient to bear in mind this slight difference, but under exceptional circumstances it is desirable to take observations in both places. Thus, in the collapse stage of cholera the rectal temperature may be several degrees above normal, while the axillary temperature may be almost as many degrees below that standard.

When it is desired to ascertain the temperature of the skin, as in studying the differences between the two sides of the body in unilateral paralysis, a special surface thermometer is employed. In one convenient form of this instrument the sensitive bulb is coiled so as to form a disc, which is applied to the skin. The reading is noted on the upright stem. The contact should be maintained for a short time only, and for the same time on the different parts compared.

There are certain diseases which run a more or less definite course, tending to spontaneous recovery, and all associated with fever, though possessing other features which enable us to distinguish one from another. These used to be recognised as the *acute specific fevers ;* they are members of the much larger class now known as *infectious diseases* or *infections*. In some of them, fever continues for a considerable number of days without any local lesion sufficient to account for it. The latter were formerly designated the *continued fevers*, and were distinguished on the one hand from the *eruptive fevers* or *exanthemata*, and on the other hand from *intermittent* and *remittent fevers*. Murchison's great work on ' The Continued Fevers of Great Britain ' recognised four continued fevers, viz., typhus, relapsing, and enteric fevers, and simple continued fever or febricula ; but this classification is now obsolete.

Very commonly, however, fever is *symptomatic—i.e.*, secondary to a local lesion, such as an abscess. In this case poisons are absorbed into the blood from the seat of disease, and cause the elevation of temperature.

Intermittent pyrexia (Fig. 1) is characterised by a febrile temperature during one portion, and by a normal or subnormal temperature during another portion of the twenty-four hours. The elevation is usually in the later part of the

day, so that the disturbance of temperature may be regarded as an exaggeration of the normal daily fluctuation. The range in disease—*e.g.*, phthisis and suppurative diseases—is commonly two or three degrees, but may be much wider.

If, with well-marked daily fluctuations, the minimum

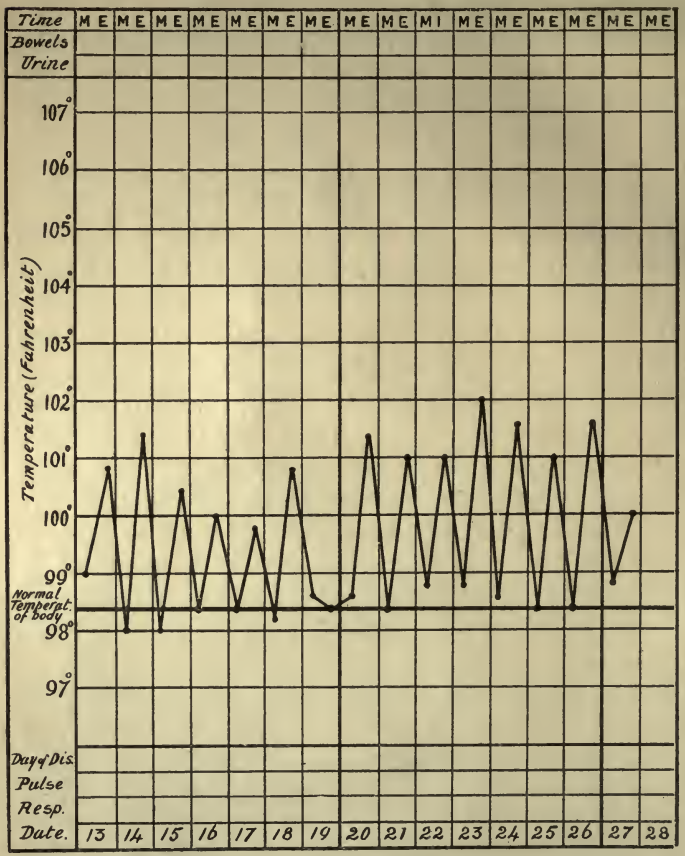

FIG. 1.—INTERMITTENT PYREXIA. (Js. L., æt. 36. Tuberculosis of mesenteric and mediastinal glands.)

temperature is higher than normal, so that the patient is never actually free from fever, the pyrexia is *remittent* (Fig. 2). Such a temperature is observed in subacute phthisis and in other diseases. The intermittent and remittent types of pyrexia are so characteristic of certain varieties of malarial

fever that the latter have long been known as intermittent and remittent fevers. *Hectic* (literally *habitual*) fever is now a popular rather than a technical expression for the fever which day after day manifests itself in the afternoon or evening, by the flush over the malar bone, and the hot skin. Hectic fever is commonly intermittent, but may be remittent; it is seen in phthisis and other chronic suppurative processes.

Sometimes an *inverse* type of temperature is observed, the

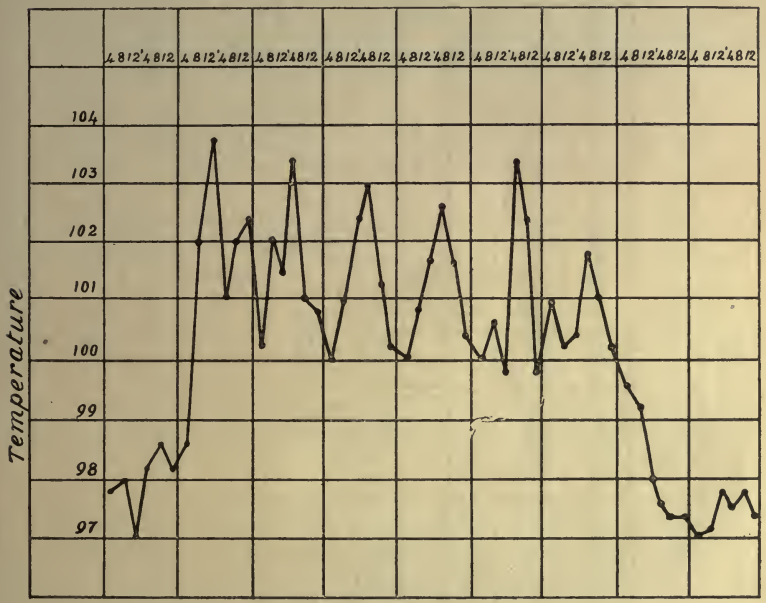

FIG. 2.—REMITTENT PYREXIA IN A CASE OF LOBAR PNEUMONIA. THE ABRUPT ONSET AND TERMINATION OF THE FEVER ARE SHOWN.

morning record being habitually higher than that of the evening (Fig. 5). This is seen in some cases of tuberculosis, and in persons who work at night and sleep during the day, as well as under other circumstances.

In some febrile diseases the onset is characterised by a rapid rise of temperature (Fig. 2). Among these may be numbered diseases beginning with severe rigors, tonsillitis, typhus, scarlatina, lobar pneumonia, etc. In others—*e.g.*, enteric, rheumatism, and lobular or catarrhal pneumonia—

the rise is gradual. In some diseases the fever comes to an abrupt end (Fig. 2). This constitutes a *crisis*, and the fall of temperature is often accompanied by other critical phenomena of a favourable kind, such as decline of the pulse-

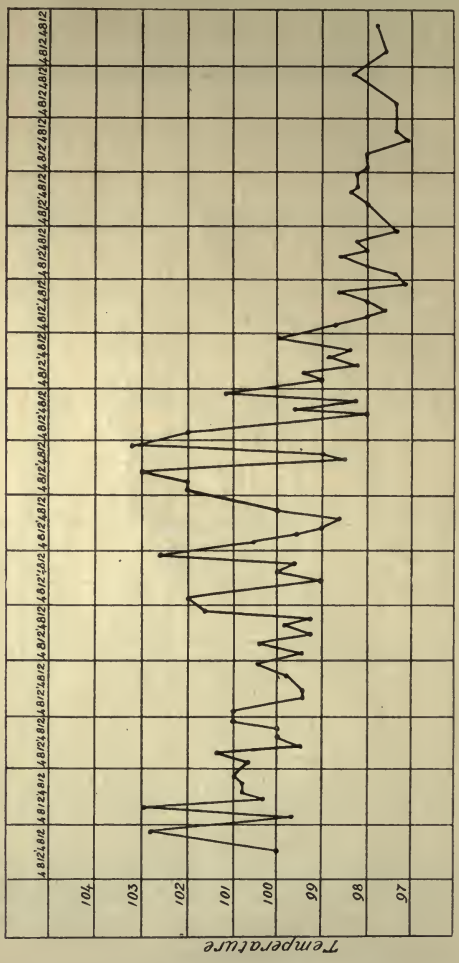

Fig. 3.—Lobular Pneumonia, with Irregular Course and Gradual Decline of the Fever.

rate, sweating, etc. This is common in lobar pneumonia, relapsing fever, and tonsillitis. Sometimes a *pseudo-crisis*, or temporary fall, is seen a day or two before the true crisis. A gradual fall, or *lysis*, is the rule in rheumatism, lobular pneumonia (Fig. 3), enteric (Fig. 4) and scarlet fever.

Very irregular and severe disturbances of temperature are met with in connection with septic diseases like pyæmia and ulcerative endocarditis.

Nature of Fever.—The heat of the body is due to the changes which take place in the tissues, chiefly the muscles, but also the glands and other organs. There is a regulating mechanism in the brain which in the healthy body keeps

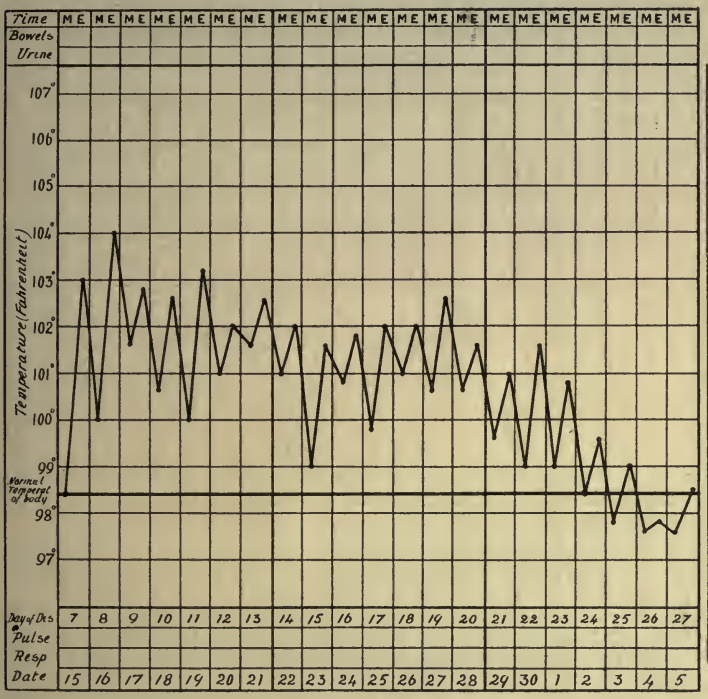

FIG. 4.—ENTERIC FEVER, SHOWING MORNING REMISSION, EVENING RISE, AND DEFERVESCENCE BY LYSIS.

the temperature almost constant. Thus, when the body-heat tends to increase through increased muscular activity, or through increased warmth of the external medium, the cutaneous vessels become relaxed, so as to permit of cooling of the blood by increased radiation ; and sweating occurs, which causes cooling by evaporation. When opposite conditions would tend to lower the body-heat, the cutaneous vessels contract to diminish the loss of heat ; and shivering

—*i.e.*, muscular contraction—may take place to increase the heat production.

Abnormal elevation of temperature results from two kinds of causes : (1) Lesions of the central nervous system (cortex, corpus striatum, crus cerebri, pons or cervical cord), which presumably either damage the regulating centre or cut off its controlling influence from the heat-producing

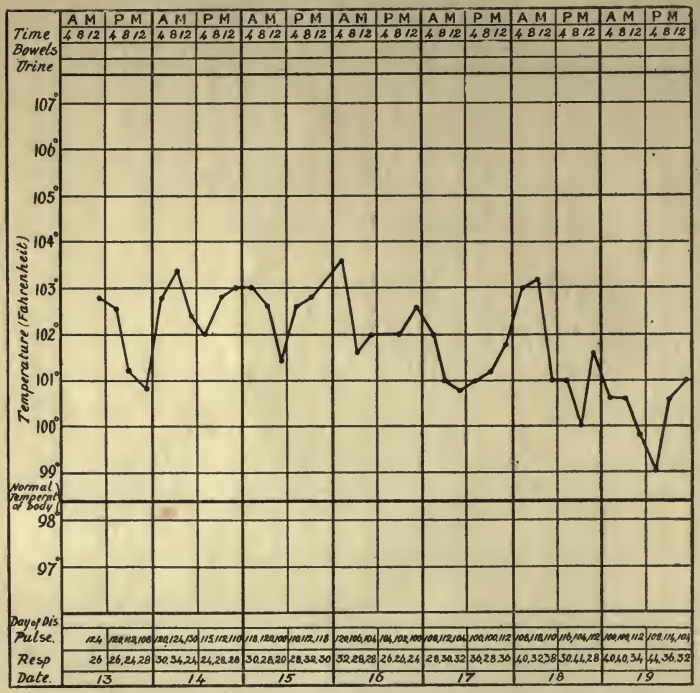

FIG. 5.—ACUTE PHTHISIS, WITH INVERSE TYPE OF TEMPERATURE : EVENING REMISSION AND MORNING RISE. (Donald M., æt. 35.)

tissues (Fig. 6) ; and (2) contamination of the blood by poisons which act upon the structures concerned in the production of heat, or upon those concerned in the removal of heat, or upon the central regulating mechanism itself. Recent researches suggest that different poisons cause fever in different ways, some increasing the production and others diminishing the loss of heat.

From what has been said, it might naturally be inferred

that fever is altogether hurtful, resulting as it does from disarranged or arrested function of important parts of the body. But the view has gained some acceptance that, so far from being an unmixed evil, pyrexia is at least protective,

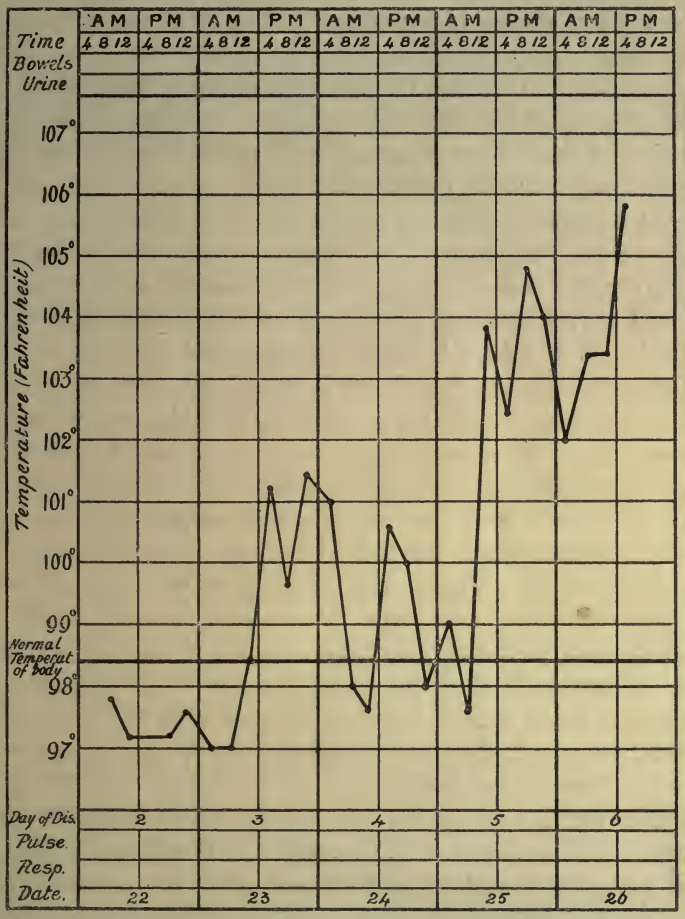

FIG. 6.—HIGH TEMPERATURE FROM SOFTENING OF LEFT CORPUS STRIATUM IN A SYPHILITIC SUBJECT. (Henry G., æt. 42.)

if not actually curative. It has been supposed that the elevated temperature favours the destruction of the poisons in the blood, and that the heat-regulating mechanism, far from being paralysed, allows the normal standard of tem-

perature to be raised for the time being to a higher figure than in health. In support of this theory it might be mentioned, first, that some organisms which at the temperature of the blood would be hurtful to man become less virulent when the temperature at which they are cultivated is elevated by a few degrees ; secondly, that certain animals, by having their temperature artificially raised, are enabled better to resist an attack of disease-producing organisms ; and, thirdly, that the diurnal variation of temperature is not abolished during fever, as might be expected if the regulating mechanism were paralysed.

Morbid Anatomy.—This is not constant in cases of fever, but one of the best recognised facts is the cloudy swelling which is often present in the cells of the muscles and glands. The heart may be dilated as a result of the degeneration of its muscle. The spleen is frequently enlarged and soft.

Treatment.—Unless the illness is very slight, the patient ought to rest in bed. In view of the great tissue waste and the tendency to exhaustion, he should get as much food as he can digest ; but as his digestive powers are usually impaired, his meals must be small and frequent, and must consist of liquids (milk diluted with soda-water or barley-water, beef-tea, soups made with vegetables but freed from solids by straining), and light farinaceous articles (arrow-root, corn-flour, oat-flour, porridge, etc.). Water, imperial drink, or lemonade may be given for the thirst, but the liquids may after a time have to be restricted on account of flatulence, which may cause undesirable upward pressure on the diaphragm and heart.

Pyrexia, being a symptom, is best treated by removal of its cause. Often, however, this cannot be done, and in any case it may be desirable, and occasionally it is absolutely necessary, to combat the abnormal elevation of temperature. Simple measures, such as cold or tepid sponging of the skin, which act by abstracting heat directly or by favouring evaporation, are always grateful, and in children may be all that is required. A simple febrifuge medicine is available in a mixture of spirit of nitrous ether and solution of the acetate of ammonium, the one ingredient causing relaxation

of vessels and the other diaphoresis. More powerful antipyretics, whose mode of action is not so clearly understood, are employed with more discrimination since it has become known that they do not cure the disease. Among these the best are phenacetin, phenazone (antipyrin), and acetanilide (antifebrin), which are often of service in relieving headache and other pains, as well as in reducing the temperature. Other drugs, such as salicin, the salicylates, and quinine, reduce the temperature in particular diseases by arresting the activity of their specific causes.

In *hyperpyrexia*, drugs cannot be relied on ; even in large doses they may exert no recognisable influence. The essential measure is a cold bath. The patient, wrapped in a single blanket, is immersed to the neck in a bath at 98° F., and the temperature of the latter is quickly reduced by cold water or ice to 65° or 60°. The patient's temperature is carefully studied, and as soon as it is reduced to 100° or 101° he is put back in bed, and wrapped in dry blankets. Stimulants should be at hand in case of a tendency to faint. If the hyperpyrexia returns, the bath must again be resorted to, and if it proves obstinate, it is well to keep the patient continuously immersed in a bath just cool enough to prevent the temperature from rising above 102°. A warm bath of this kind is not nearly so likely to cause contraction of the superficial arterioles as a cold bath, and it thus helps to ward off two risks, since contraction of the arterioles would (1) greatly diminish the quantity of blood which could be cooled by the agency of the bath, and (2) greatly increase the strain upon the heart, which is already seriously weakened by the febrile condition. The accompanying chart (Fig. 7, p. 12) shows the influence of cold and of continuous warm baths on the temperature in a case of rheumatism, with double pneumonia and hyperpyrexia.

INFECTIOUS DISEASES.

The expression 'infectious diseases' is much more comprehensive than it was a quarter of a century ago, chiefly because many diseases have been brought into this group

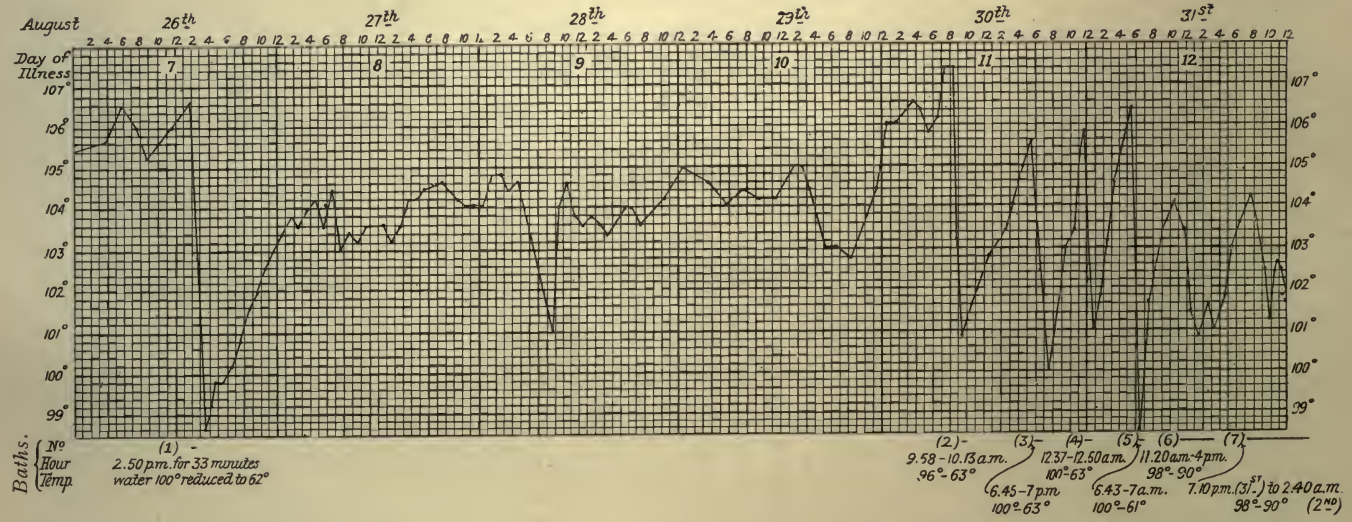

FIG. 7.—HYPERPYREXIA IN RHEUMATIC POLYARTHRITIS WITH DOUBLE PNEUMONIA. INFLUENCE OF COLD AND WARM BATHS SHOWN. DURATION OF PYREXIA THIRTY-EIGHT DAYS. (John G., æt. 30.)

which formerly were classified otherwise. And, indeed, the meaning of the term *infectious* has undergone some modification in connection with this change. Formerly the word was applied to diseases which were transmissible, either by direct contact of person with person, or indirectly by air breathed, water or food swallowed, or otherwise. The word *contagious* was generally used in exactly the same sense, but sometimes in a more literal and restricted sense, being applied to diseases spread by contact of person with person (*e.g.*, syphilis, gonorrhœa), or, at least, communicated in a less subtle way than by the medium of respiration. *Infective*, again, was applied to diseases which, having gained a footing in the body, tended to spread from the seat of invasion to other parts of the body. It was thus used of tuberculosis and syphilis, but with less frankness of malignant growths. Of late, however, the tendency has been to avoid the use of the word *contagious* in scientific writings, and to regard *infective* and *infectious* as synonymous. Sometimes the infections are divided into two classes, the *transmissible* and the *non-transmissible*, according as they are or are not communicable from the affected to an unaffected individual ; but as the modes of transmission present all degrees of directness and indirectness, this classification is no improvement on the other.

An infection which is constantly prevalent in a locality is said to be *endemic* in that locality. A disease which rapidly attacks a large number of persons in a locality is spoken of as *epidemic*. An endemic disease may from time to time break out in epidemic form (*e.g.*, scarlet fever or measles, in large cities).

Most infectious diseases are caused by bacteria, but a few (*e.g.*, malarial fever) are caused by lowly forms of animal life. It is this fact of causation by one of these lowly vegetable or animal organisms which for the present gives to any disease a title to be called *infectious*. Curiously enough, a large proportion of the diseases to which the word was originally applied (measles, whooping-cough, typhus, scarlet fever, etc.) have as yet had no specific micro-organism allotted to them. The modern nosologist permits them

to remain, meantime, among the infections, because their clinical course and their communicability (their 'infectiousness' in the original sense of the word) make him think of such a cause as likely. We are apt to speak of the infections as caused by *specific* micro-organisms—the idea being that each infection is related to a micro-organism of a particular species. This is by no means always the case. Thus, on the one hand, endocarditis may be caused by different species of microbes; and, on the other hand, the pneumococcus may cause not only pneumonia, but also meningitis, otitis media, endocarditis, pericarditis, and pleurisy.

Many of the infections run a more or less definite course, with a tendency to end in spontaneous recovery, though death often takes place from exhaustion or from some complication. In a number of diseases of this group it is possible to recognise five stages—viz., (1) *incubation*, (2) *invasion*, (3) *eruption*, (4) *defervescence*, and (5) *convalescence*. 1. In the first stage, the virus, having already obtained access to the body, multiplies for a time without causing definite symptoms. 2. The invasion is characterised by a rise of temperature and various other symptoms, some of which are common to all febrile attacks, whilst others are more characteristic of the particular disease. 3. In the third stage there is frequently a characteristic cutaneous eruption, but sometimes there is continuous fever without any rash. 4. In the fourth stage, the disease is dying out, and the temperature falls, in some cases by crisis and in others by lysis. 5. In the fifth stage the disease proper has passed away, and the patient is recovering, or still suffering from its effects, whether this be mere weakness or some more serious sequel of the infection. Apart from diseases such as malarial fever and relapsing fever, where intermissions and relapses are a characteristic feature, and apart from the occurrence of complications, it sometimes happens that the disease starts afresh after apparently coming to an end; and sometimes after it has declined, though without obviously reaching its end, an exacerbation occurs. Such a fresh start is called a *relapse*, and such an exacerbation is

called a *recrudescence*. Both are well known in enteric fever.

Whilst in some infectious diseases—*e.g.*, septicæmia—the micro-organism is regularly present in the blood from the time of invasion, in others—*e.g.*, erysipelas—it begins in a limited area of the tissues, from which it may quickly or slowly spread to other parts, though not entering the blood in any quantity sufficient to cause the symptoms of a general febrile disease. In the long-run, however, it may enter the blood in quantity—*e.g.*, in tuberculosis. In yet other instances—*e.g.*, tetanus—the micro-organism remains throughout at the seat of entrance, and does harm to the patient only through the absorption of its products.

Mixed infections are frequent, and the severity of an infection is often greatly augmented by the simultaneous presence of one of a different kind (as in tetanus with suppuration). Frequently, however, when an individual suffers from two such invasions, the one is secondary to the other—*e.g.*, whooping-cough occurring after an attack of measles, or suppurative processes occurring secondarily to various eruptive fevers.

Immunity.—Certain individuals suffer more readily than others from infectious diseases, and such special susceptibility on the one hand, or immunity on the other, may, as is well known, prevail in families. With regard to certain diseases, this peculiarity may characterise a race. But the most important immunity against an infectious disease is that which is conferred by an attack of the disease itself. Like every other kind of immunity, however, this is a merely relative one ; for if, through defect of food or clothing, want of rest, overwork, or other unsatisfactory conditions of life, the general vigour of the individual be temporarily lessened, he may fall a victim to a disease which, under normal circumstances, he would assuredly have escaped. The degree of immunity varies much with the disease as well as with the individual. Thus, whooping-cough and chicken-pox rarely attack an individual a second time ; with several other fevers a second attack is unusual, though not rare ; whereas

one attack of pneumonia seems actually to predispose to a later attack.

The symptoms of an infectious disease are due, in great part, to the action of the micro-organisms and their products on the tissues, but also in part to the abnormal activity of the tissue-cells. These soon succumb if the attack of the parasitic organisms is extremely severe ; but if it is short of this, an energy, usually dormant or sluggish, wakes up in the tissue-cells, and they give rise to substances (*antisubstances*) which are antagonistic to the invaders. Whether the latter be bacteria, chemical products of bacteria, foreign blood corpuscles, or any other foreign cells or cell products, the healthy animal body produces a substance (*antisubstance*) which is *specifically* antagonistic to that particular kind of intruder.

These antisubstances are not to be regarded as entirely new productions on the part of the body, but as molecules which are naturally present in the fluids or tissues. When a foreign cell, such as a bacterium, invades the body, it soon neutralises the already existing molecules of the antisubstance with which it has specific affinity ; but this, under favourable circumstances, stimulates the tissue-cells to produce that particular antisubstance in great abundance, with the result that the antisubstance comes to be present in the body in excess of the immediate requirements, and so immunity is induced. Immunity acquired in this way is described as *active*, because it results from the vital activity of the tissue-cells of the person or animal. *Vaccination* or *preventive inoculation* aims at establishing this variety of immunity, and is extensively employed to secure protection against small-pox, hydrophobia, enteric fever, plague, and cholera.

When serum from an animal which has acquired an active immunity against a certain disease is introduced into the body of another animal, the latter will be rendered immune to that particular disease. Immunity acquired in this way is described as *passive*, because it does not depend upon the vital activity of the cells of the animal so rendered immune. It is a more transient phenomenon than active

immunity. The production of passive immunity is the aim of *serum-therapeutics*.

An antisubstance neutralises the invading agent with which it has a specific affinity by entering into chemical combination with it. In the case of tetanus and diphtheria this seems to represent the whole effect which the antisubstance has upon the attacking agent. In each of these diseases the symptoms are due to absorption of the soluble poison or toxin of the microbes, and the tissue-cells secrete into the fluids around them an antitoxin which is specific to the particular toxin. Thus the antitoxin of diphtheria unites chemically with the toxin of that disease, and if produced in sufficient quantity, prevents the toxin from uniting with the tissue cells and poisoning them. In the same way the antitoxin of tetanus will neutralise the toxin of that disease. The serum of animals artificially immunised by repeated injections of the toxin of one or other of these diseases contains the respective antitoxin, and is employed in the treatment of patients (*antitoxic serum*).

In the case of most infections, however, the specific microbes do not secrete powerful soluble toxins which might be employed to evolve corresponding antitoxins in animals, and thus provide us with curative antitoxic sera. The poisons in those diseases (tuberculosis, enteric fever, cholera, pyogenic micrococcus infections, pneumococcus infections, etc.) appear to be largely 'intracellular poisons,' in the sense of being contained within the bacteria themselves, and only liberated when the microbes degenerate or die. The immunity that arises in such diseases, whether they are the result of artificial inoculations or are ordinary spontaneous attacks, is due, not to a single substance like antitoxin, but to a bacteriolytic or bactericidal agency which consists of two elements. One of these is the specific antisubstance developed in the course of the immunising process, and known as the *immune-body*. The other is normally present in the serum or tissues, and is known as the *complement* or *alexin*. It has been supposed that the immune-body operates by linking itself on the one hand to the bacterium, and on the other hand to the complement, thus enabling the latter to

destroy the former. Unfortunately the complement is not increased by the immunising process, and it is readily destroyed (*e.g.*, when outside the body, by keeping). An antibacterial or antimicrobic serum is prepared by injecting into animals increasing doses of cultures containing the specific micro-organism. Such a serum, though damaging to the particular microbe used in its preparation, does not protect against the essential toxin of the microbe, and is therefore of no great curative value, though it may be useful in prophylaxis. Some antitoxic sera, on the other hand, possess antimicrobic in addition to their antitoxic properties. Among the antibacterial sera which have been prepared are the antityphoid, anticholera, antiplague, antipneumococcic, and antistreptococcic.

Some antimicrobic or antibacterial sera, when added to living cultures of the particular organisms with which they have specific affinity, cause the bacteria to clump together. This clumping or agglutination is due to an antisubstance known as an *agglutinin*, which is present in the serum. Whether the agglutinin and the immune-body in such a serum are one and the same antisubstance is still a matter of speculation; there is some reason to believe that they are not identical. Agglutination is met with in connection with enteric, cholera, Malta fever, glanders, plague, and bacterial dysentery. It is often taken advantage of for diagnostic purposes, as in Widal's test for enteric fever.

The special activity of the tissue-cells which has been alluded to, and which manifests itself by the production of antisubstances as well as in other ways, is spoken of as their *reaction* to the assault of the bacteria. The defervescence of a fever will naturally set in as the tissues become able to produce a sufficiency of the antisubstance to neutralise the toxin or render harmless the bacteria in the body. As long as the tissue-cells keep up this activity, so long may the immunity be expected to continue. The bacteria present in the body, but now rendered harmless by the neutralisation of their products, or by inhibitory or destructive effects upon themselves, are readily disposed of by phagocytes, which ingest and digest them. Wright, indeed, has recently de-

scribed, under the name of *opsonins*,[1] bodies which are present in the normal serum, and which have the property, not of destroying bacteria themselves, but of rendering them a more easy prey to phagocytes. When a particular microbe is injected into an animal's body, the specific opsonin for that microbe becomes more abundant in the serum.

The facts and hypotheses that have been adduced here on the subject of immunity are due in great measure to Ehrlich and his followers, and it is easy to infer from them, as is now generally done, that phagocytosis is an evidence that immunity already exists, rather than itself the explanation of immunity. According to Metchnikoff, however, such an inference entirely fails to do justice to the importance of phagocytosis, which he holds to be the one constant factor in immunity, whether natural or acquired. Metchnikoff recognises two kinds of phagocytes. (1) *Macrophages* are large non-granular cells which occur both in the circulation (large lymphocytes) and in the fixed tissues. They devour cells of animal origin such as red blood corpuscles; lowly animal parasites, such as trypanosomes and the parasites of malaria; and the microbes of chronic infections, such as tuberculosis and leprosy. Their power of digesting these foreign cells depends upon a cytase (*macrocytase*) or soluble ferment (corresponding to a complement or alexin), which is contained within the phagocytes, and is not liberated into the serum unless the phagocytes are injured. (2) *Microphages* are the granular polymorphonuclear leucocytes of the blood, whose granules may be either neutrophile or eosinophile. They attack the microbes of the acute infections, and digest them by means of their intracellular cytase (*microcytase*). These two kinds of phagocytes, then, with their cytases, account for natural immunity, according to Metchnikoff.

In connection with acquired immunity, however, an additional kind of soluble ferment is recognisable, viz., a *fixative* (corresponding to an opsonin), which, though produced by the phagocytes, passes freely into the serum. This fixative does not itself kill the bacteria, but it attacks

[1] Greek, ὀψώνης, a purveyor.

them in such a way as to make them become a more easy prey to the cytase. The fixatives are mostly specific to the particular bacterium or other foreign cell which, by invading the body, has led to their development in quantity; whereas the cytases are not specific.

It will be seen that Metchnikoff, the original expounder of the older phagocytic theory, attaches most importance in immunity to living cells which contain soluble ferments in their substance; whereas his opponents, who hold the more recent humoral theory, attach most importance to non-living secretions which exist free in the serum.

The supply of phagocytes depends, of course, on the activity of the bone-marrow and lymphatic tissues, and in such a disease as pneumonia, where a well-marked leucocytosis is the rule, the non-appearance of this sign of a vigorous reaction on the part of the phagocyte-forming tissues is a fact of evil omen.

1. TYPHUS FEVER

(Exanthematic Typhus. Malignant Fever. Petechial Fever. Putrid Fever. Spotted Fever. Gaol, Camp, Ship, or Hospital Fever).

Definition.—A specific infectious fever, fostered by bad hygienic conditions; setting in suddenly; associated with a cutaneous eruption which is partly petechial, and with severe nervous prostration; and terminating by crisis about the fourteenth day.

Etiology.—The specific microbe, if there be one, has not yet been demonstrated with certainty. The disease spreads by direct contact with the sick and by fomites.[1] The one condition which, above all others, acts as a predisposing cause, is overcrowding in ill-ventilated dwelling-places. Some of the older names of the disease, quoted above, indicate only too truly the diversity of circumstances under which it was liable to break out. Medical men still in practice had, as a matter of course, to run the gauntlet of

[1] Fomites are articles which have been in contact with the patient.

typhus fever when they were attending the general hospitals as students, and large numbers of them took the disease. Within more recent times the disease has been epidemic at intervals in our large towns, but for some years past it has been nearly obsolete in this country. The virus seems to acquire great intensity in ill-ventilated slum-dwellings, but when the patient is removed to the airy wards of a modern fever hospital, it rapidly loses this character, and the student may then inspect the case with practically no risk to himself. Poverty and exhaustion may predispose by lowering the resisting power. As with pneumonia, so in the case of typhus, habitual drunkenness seems to render the individual more susceptible to the infection, as well as more likely to succumb to the attack. The fact that typhus is most common in winter is to be explained by the greater crowding within doors, and the smaller amount of fresh air admitted by open windows and doors at that season. The most susceptible age is between ten and twenty.

Morbid Anatomy.—There is no characteristic lesion. The appearances are almost solely those of severe pyrexia. The petechiæ persist after death. The blood is dark and liquid. The heart is soft, and its muscular fibres are granular. There is hypostatic engorgement of the lungs. The spleen is large and soft, and there is cloudy swelling of the liver and kidneys.

Incubation.—The incubation period is usually about twelve days. It is seldom longer, but occasionally much shorter.

Symptoms.—The invasion is usually sudden, and is characterised by recurring attacks of chilliness or distinct rigors; headache, generally frontal, and sometimes intense; pains in the back and limbs; a sense of prostration; thirst, anorexia, sickness, and sometimes vomiting; constipation and a furred tongue; a flushed face, bloodshot eyes, and contracted pupils.

The temperature rises quickly, and may reach or exceed 103° on the first evening. It continues to show an upward tendency for a few days longer. The characteristic rash appears about the fifth day (fourth to seventh), and this

'mulberry rash' consists of two elements—namely, spots and mottling. The spots are small, pink, ill-defined, and slightly elevated. When fresh, they disappear on pressure. They are generally first seen on the anterior folds of the axillæ, but they soon spread over the trunk and limbs. They are usually abundant on the back of the trunk and backs of the hands, but are seldom well marked on the face. After a time the spots become the seat of minute hæmorrhages (petechiæ), and accordingly cease to be removable by pressure. They persist till the crisis. The second element in the rash is a dusky marbling ('subcuticular mottling').

In the latter part of the first week, the pulse is still becoming more rapid and feeble, the bodily strength is diminishing, the tongue is dry and brown, and the temperature continues high (103° to 105°), with but a small diurnal variation. There may be some delirium at night. There is sometimes a peculiar odour about the patient's body.

In the second week, the symptoms attain their fullest and most characteristic development. Headache is replaced by delirium, which is occasionally violent, but more frequently of a quieter, talkative kind. The patient lies on his back, deaf, and insensible to everything around. The eyes may be closed, but sometimes they are open, and this condition, if associated with muttering, suggests that the patient is awake, while he is really almost comatose ('coma vigil'[1]). There is usually retention of urine, but the evacuations are often passed into the bed unconsciously. The pulse is rapid, soft, and sometimes irregular. The first sound of the heart may be almost inaudible at the apex. The breathing is rapid. The tongue is dry and brown, and the lips and teeth are foul with sordes. Other symptoms of the intense prostration are carphology, floccitation, and subsultus tendinum.[2]

[1] *Coma* (κῶμα), deep sleep; *vigil*, awake.

[2] Carphology (κάρφος, chaff; λέγειν, to collect) means grasping at imaginary objects, but is sometimes used in the same sense as floccitation. Floccitation, or floccillation (*floccus*, a flock of wool), means picking at the bedclothes. *Subsultus tendinum* means jumping or jerking of the tendons, resulting, of course, from muscular contractions. These and other symptoms of typhus in the second week are phenomena of the so-called 'typhoid state.'

The stupor may deepen to fatal coma, or the heart may fail, but in most cases a change for the better sets in about the fourteenth day. The temperature falls by crisis, the pulse becomes stronger and less frequent, the skin becomes moist, and the patient sleeps more naturally. He soon becomes free from head symptoms, and has a keen appetite, but is for a time intensely feeble.

Varieties.—Sometimes the disease runs a very mild course, and ends by an early crisis (*typhus febricula*). In epidemics occurring in time of war, the disease has sometimes proved fatal in a day or two (*typhus siderans*). Another severe form is associated with hæmorrhages from various organs.

Complications and Sequels.—Among the most important are bronchitis, pneumonia, hypostatic congestion of the lungs, pleurisy, uræmia, thrombosis of arteries and veins in the limbs, bedsores, glandular swellings, abscesses, paralysis and dementia. A certain amount of bronchial catarrh is almost normal to the disease. The severe congestion and œdema of the bases of the lungs, known as hypostatic engorgement, are attributable to the enfeeblement of the heart. Arterial obstruction may give rise to gangrene of the extremities, external genitals, etc. Thrombosis of the femoral vein may cause 'white leg' or 'swelled leg' (phlegmasia dolens), a condition characterised by pain and great swelling of the affected limb. This usually subsides in the course of some weeks, though for long afterwards œdema may continue to appear about the foot when the patient walks.

Diagnosis—*From Enteric Fever.*—In typhus the sudden onset, the rapid rise of temperature, the slight morning remission, the defervescence by crisis, the duration for two weeks, the earlier appearance of the eruption, the ultimate fixation of the spots, their development in one crop and their abundance on the backs of the hands and feet, the mottling of the skin, the brown, crusted tongue, the absence of diarrhœa and other abdominal symptoms, the small pupils, and a history of contagion, are important evidence.

From Measles.—In measles there are sneezing, watering of the eyes, and a peculiar cough in the catarrhal stage. The rash appears on the fourth day, comes out strongly on

the face, and when fully developed is quite different from that of typhus. Moreover, the temperature begins to fall within two days after the rash first appears. There may be a history of contagion.

From Purpura.—Typhus is distinguished by the course of the temperature, and by its epidemic incidence.

From Uræmia.—Typhus is distinguished by the temperature. Apart from convulsions and other recognisable complications, the temperature in uræmia is generally below rather than above the normal. The history of the case in chronic uræmia, and the condition of the urine in acute kidney disease, give important information.

From Bronchopneumonia.—The latter is very commonly secondary to some other disease, such as measles, whooping-cough, or bronchitis. Its onset is often less sudden than that of typhus ; its course is variable and often protracted. There is no rash. The physical signs in the chest may be of diagnostic value.

From Meningitis.—In meningitis the temperature and pulse are quite different from those of typhus. Vomiting is more constant. Delirium, if present, is added to, instead of replacing the headache. The cranial nerves are likely to be involved. There is no rash as a rule. A primary lesion is often present elsewhere—*e.g.*, pulmonary tuberculosis or pneumonia.

Prognosis.—In the period between five years of age and middle life the prospect of recovery is almost inversely as the age of the patient. Under twenty, the mortality scarcely amounts to 4 per cent. In the first three years of life, however, the mortality is specially high. After middle life, it exceeds 50 per cent. The outlook is unfavourably influenced by preceding fatigue, by alcoholism, and by the early occurrence of intense prostration. Irregularity or slowness of the pulse, marked hypostatic congestion, and marked ' typhoid ' phenomena, are also ominous.

A second attack is rare.

Treatment.—The patient should be kept in a large airy room, and should be attended night and day by experienced nurses. Different beds for day and night are desirable.

Liquid food should be given ; for an adult, say 4 ounces every two hours. Milk is most to be relied upon, but beef-tea, egg-flip, and meat-jelly may also be administered. Cold water should be given freely as a drink. The bladder must be carefully attended to. The mouth must be frequently cleansed, and the skin should be regularly sponged with antiseptic preparations. The position of the patient should be changed from time to time, so as to diminish the risk of severe hypostatic engorgement. In young subjects and in mild attacks alcohol need not be given as a matter of routine, but in a large proportion of cases stimulants of some kind are required sooner or later. Among those that may be recommended are camphor (2-grain doses in milk), spirit of ether ($\frac{1}{2}$-drachm doses), and ammonium carbonate (5-grain doses).

Severe headache and insomnia may be treated by the local application of ice, or by the internal administration of citrate of caffeine or of morphine. For violent delirium, ice may be applied to the head, or the patient may be put in a cold pack. For constipation, castor oil ; and for diarrhœa, lead and opium pill may be given. Cardiac failure should be treated by strychnine and ammonia, and the same remedies are indicated in hypostatic congestion. In the latter case, large poultices should be applied to the chest. Before the patient is allowed to mix with others, he should have several baths and should have his clothes thoroughly disinfected.

The period of isolation should extend to five weeks from the commencement of the illness.

2. ENTERIC FEVER[1]

(Typhoid Fever. Gastric Fever. Nervous Fever. Abdominal Typhus. Pythogenic Fever. Febricula. Simple Continued Fever. Infantile Remittent Fever).

Definition.—An infectious fever due to a specific microbe, and characterised by a special lesion of the intestine, by

[1] The disease known in the mountainous regions of the Western United States as *mountain fever* has been shown to be enteric fever.

swelling of the mesenteric glands and spleen, and by a rose-coloured rash.

Etiology. — The specific microbe is Eberth's bacillus (*Bacillus typhosus*), a rod with rounded ends, about 3 μ long and 0·5 μ thick, possessed of numerous flagella, and in liquid media actively motile. It is stained by the ordinary aniline dyes, but is decolourised by Gram's method. It can live in fresh and salt water, in shell-fish such as oysters, and in dry earth and sand. It can multiply in milk and butter. The evidence available goes to show that the bacillus invades the body from the lumen of the intestine after being swallowed, and not by way of the blood. In cases of enteric, the bacillus is present in the inflamed follicles of the intestine, and in the mesenteric glands and spleen ; but to a smaller extent it obtains access to the blood and distant organs. It is usually present in the fæces at some stage, frequently in the urine, and sometimes in the sputum.

It would appear that the bacillus is often not fully virulent until some period of time after its evacuation in the stools or urine of the patient. The development of its virulent characters is favoured by stagnation of the water in which it is present, by warmth, and by scantiness of sunlight and fresh air, whereas it is retarded or prevented by the opposite conditions. Thus, a hot, dry summer, which favours stagnation in the sewers, is apt to be followed by an autumnal outbreak of enteric in the large towns, where a certain amount of enteric excreta is always present in the drains.

Infection takes place through swallowing of the bacillus, which leaves the patient by the fæces and urine. The most common vehicle in European countries is water contaminated by sewage from a case of the disease, but in hot countries dust seems to take the place of water for this purpose. The water may be used for drinking, or for adding to milk, or for washing utensils in which milk is kept. Milk and its derivatives (butter, ices, etc.) may be contaminated in the ways indicated, or by being taken from the cow, or otherwise handled, by a person who is nursing a case of enteric, or who is actually suffering from the disease. Shell-fish and vegetables which are eaten raw may be contaminated,

and so spread the disease. Flies may convey the infection from typhoid stools to food. The disease is not contagious in the literal sense of the word, and it does not readily spread from the patient to those in attendance. Nevertheless, young nurses in charge of enteric cases frequently acquire the disease. This is doubtless owing in most cases to lack of care in cleansing the hands, but it is probable that the infection is sometimes aerial ; portions of the excreta have dried on the bedclothes, and then been blown about, so that some bacilli may have reached the pharynx of the nurse. Occasionally gas arising from a drain or water-closet appears to be the vehicle of infection. This may carry the bacilli from the urine, fæces, or soiled bedclothes of the patient to the food or drink of the healthy.

Enteric fever is a disease chiefly of childhood and early adult life. It prevails more or less in all parts of the world, but is liable to assume a local epidemic form in the presence of imperfect sanitary conditions. Autumn is the season when most cases are met with. Individuals and families differ greatly in their liability to this disease. Mental depression, physical debility, chill, and influenza are among the predisposing causes.

Morbid Anatomy.—Hyperæmia and proliferation of the lymphocytes occur in the *Peyer's patches* and *solitary follicles* of the ileum, which thus become pink and swollen. About the tenth day of the disease this process begins to give place to necrosis, so that the patches and follicles are converted into sloughs, which are often stained yellowish-brown by the bile-pigments. In the third week of the disease, the sloughs are separating, leaving the typhoid ulcers with their edges undermined. A patch may separate in one piece or in several fragments. Sometimes it presents for a time a worm-eaten appearance, owing to the development of numerous foci of ulceration, which afterwards spread and coalesce. The floor of the ulcer may be the muscular coat or the peritoneum ; or the whole thickness of the intestinal wall may be involved in the necrosis. The ulcer usually heals in the course of a week or ten days, but sometimes this is postponed for several weeks ; while a third possibility is that perforation may take

place. As already indicated, the original slough may include the whole thickness of the wall, but sometimes, when the original depth is less, ulceration gradually extends through the peritoneal wall; and, thirdly, it is probable that the peritoneal coat is sometimes torn by movements of the bowel or otherwise. In mild cases, the inflammation may subside without necrosis or ulceration.

The solitary follicles of the large intestine may share in the morbid process.

The *mesenteric glands* are swollen and firm through infiltration. The *spleen* is enlarged, and usually, but not always, soft. Both contain the specific microbe. The bone-marrow shows changes similar to those in the lymphoid tissue.

The *lungs* may be congested or actually inflamed, the *larynx* ulcerated, the *heart muscle* degenerated, and the *voluntary muscles* (especially of the abdomen and thighs) in a state of coagulation necrosis (Zenker's 'vitreous degeneration'). The cells of the *liver* and *kidneys* are also changed in consequence of the toxic and febrile state.

Incubation.—The incubation period is about twelve days (two to twenty-three days).[1] In some cases the patient feels out of sorts at this stage.

Symptoms.—The features of an attack of typhoid fever vary to an extraordinary extent in different cases. The onset is usually gradual, the patient suffering at first from chilliness, lassitude, anorexia, and headache. There may be epistaxis, and the gradual rise of temperature is often associated with some abdominal discomfort and diarrhœa. In most cases the patient is not forced to take to bed for some days after the onset, and he sometimes keeps on duty for the first week or two, or even ('ambulant' or 'ambulatory' type) throughout the entire illness.

The temperature continues to rise during the first week. In a typical case it shows a rise of from 1° to 2° each evening, and a fall of less than this on the following

[1] A nurse in Paris swallowed an ounce of a broth culture of Eberth's bacillus with suicidal intent. Headache began on the third day, and she passed through a severe attack of the disease (*Med. Press*, September 21, 1904, pp. 316, 317).

morning ; and so on, until the pyrexia attains an elevation of perhaps 103°.

By the end of the week the symptoms are usually characteristic. The temperature shows a well-marked morning remission. The pulse, at first bounding, is now soft and full, and has a rate of about 100 per minute. The tongue is moist, red at the margins, and covered over the dorsum with a yellowish fur. There is usually diarrhœa, the bowels being moved several times daily, and the motions resembling pea-soup in appearance. Often, however, there is constipation, either all through, or with the exception of a day or two. The abdomen is somewhat distended, and there may be some gurgling and tenderness in the right iliac fossa. The splenic dulness is increased, and the enlarged spleen can often be felt. The cheeks are flushed and the pupils are of large size. Sweating is not uncommon. Sometimes a scarlatiniform rash appears at this time and introduces an element of doubt as to the diagnosis.

The characteristic eruption appears about the seventh day ; sometimes earlier, sometimes later, sometimes not at all. The little oval rose-coloured spots (' rose papules,' ' lenticular spots '), being slightly elevated, can be felt by the finger, and are influenced by pressure all through their existence. The rose-spot has sometimes a minute vesicle on its top. The spots come out in successive crops, and are found chiefly on the trunk and shoulders. A dozen may be found at one time in an ordinary case, but their number may be much smaller, and occasionally they are present in extraordinary profusion. Some significance was formerly attached to the *taches bleuâtres* which are sometimes recognisable on the skin. These are faint bluish marks in the same plane as the skin itself. They are not, however, confined to this disease, and are probably due to pediculi.

In the second week, and still more in the third, the general symptoms are intensified. The patient is emaciated and feeble. He may be deaf. The cheeks are flushed. The tongue is dry, and either brown, or clean, red, and fissured (' beefy '). The pulse is very soft, and ranges from 110 upwards. The red corpuscles and hæmoglobin of the blood

are diminished. The white corpuscles are also as a rule reduced in uncomplicated cases ('leucopenia'). The abdomen is prominent. Diarrhœa is severe. The stools are offensive, and in the later stages may contain sloughs or blood. Delirium is common, especially at night, and in severe cases the 'typhoid state' may supervene, with hypostatic congestion or inflammation of the lungs. The temperature commonly shows a distinct morning remission.

About the end of the third week, defervescence generally sets in, and the symptoms become less urgent. The temperature falls by lysis in such a way that the pyrexia is for a time remittent, and thereafter for a time intermittent; each morning fall exceeds the previous evening rise until the normal is attained, usually in the fourth week.

Varieties.—The onset may be sudden, and in some of these cases head symptoms appear early. Or the disease may terminate by crisis in the second week (*abortive* type). Very mild cases with trivial symptoms may have the morning temperature nearly normal (*enteric febricula*). Such cases may be 'ambulant' until possibly a fatal perforation occurs. Occasionally the temperature is normal throughout. In some instances, the fever is much more protracted than has been indicated, and four or five weeks may elapse before defervescence commences (see Fig. 8). The normal or subnormal temperature which follows the lysis is readily disturbed by slight causes, of which indiscretion in diet is one of the most important.

Relapses.—These are not uncommon, and are characterised by the various phenomena of the original attack, including the eruption. They often set in about the tenth day after the primary attack ceases, and are attributable to a fresh infection of the intestine. They are usually milder and briefer than the first attack, but in exceptional instances they terminate fatally. Occasionally two or three relapses occur. Recrudescences ('intercurrent relapses') are to be distinguished from relapses; they are exacerbations in the course of the original disease, and are attributable to extensions of the lesion (Fig. 8).

Complications and Sequels.—*Intestinal hæmorrhage* is one

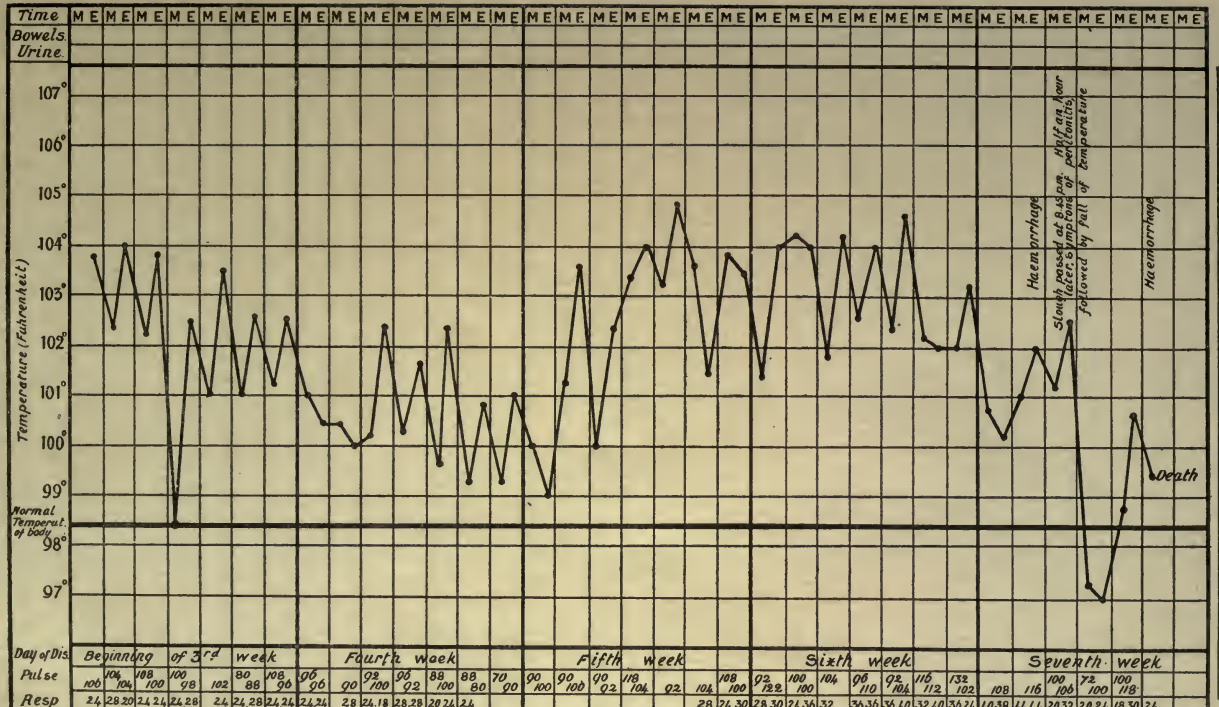

FIG. 8.—ENTERIC FEVER, WITH RECRUDESCENCE IN FIFTH WEEK, AND DEATH IN SEVENTH WEEK. (Thos. H., æt. 18.)

of the most formidable complications, and occurs chiefly in the third and fourth weeks, when the sloughs are separating, but sometimes later. The loss of blood may be trifling, or it may be so great as to cause immediate death. The blood may be bright red, or clotted, or mixed with fæces (the stools then resembling strawberry jam), or tarry. If the bleeding is severe, it causes a sudden fall of temperature and symptoms of collapse.

Perforation is another dreaded complication. It may supervene as the sloughs are separating in the third and fourth weeks, or later if the ulcers become chronic. It is indicated by sudden abdominal pain and other symptoms and signs of general peritonitis, including a sudden development of the Hippocratic facies, an increase in the pulse-rate, an increased frequency and thoracic type of respiration, a fall succeeded by a rise of temperature, rigidity of the abdominal wall, and in the course of a few hours signs pointing to the presence of fluid in the abdominal cavity.[1] There is frequently, but not always, a leucocytosis. Death takes place within two days. Occasionally, however, a patient recovers after manifesting symptoms of perforation; this is to be explained by the ulceration not extending completely through the serous coat, or by its effects being limited by adhesions.

Tympanites, or *meteorism*, is an exaggeration of the abdominal distension which is normal to the disease. When it is severe, it is not only annoying to the patient, but it tends to restrain the activity of the diaphragm and thus interfere with respiration, and also to embarrass the cardiac action. Moreover, the tension exerted upon the wall of the intestine favours the occurrence of perforation.

Bronchitis is very common in enteric. In severe cases hypostatic congestion of the lungs is frequently met with; it is pointed to by great rapidity of breathing, with impaired

[1] General peritonitis is occasionally met with in enteric fever where no perforation can be detected. This may be due to pyogenic organisms working their way through the base of an ulcer; to infection by the blood; or to suppuration and rupture of a mesenteric gland, of the spleen, or of the gall-bladder.

percussion, and fine, moist râles at the posterior bases. Lobar and lobular pneumonia, pleurisy, and ulceration of the larynx, are other complications connected with the respiratory system, whilst empyema and phthisis pulmonalis are occasional sequels.

Thrombosis of a vein, usually the left femoral, is common late in the fever or in convalescence (marasmic or marantic thrombosis, phlegmasia dolens, phlegmasia alba dolens, white leg). The limb swells greatly, the skin becomes tense and shiny, and pits slightly on firm pressure, and the obstructed vessel may be felt as a firm, tender cord. In the course of some weeks the circulation is restored, but for a long time afterwards the foot is apt to swell when the person walks much. Bradycardia and tachycardia are occasional sequels of enteric.

Retention of urine is well known in severe cases, but it may be present quite early in a mild attack. The urine is often albuminous in consequence of the febrile state, but sometimes acute nephritis occurs. In some instances the bacilli are present in the urine in such numbers as to render it turbid, and yet without necessarily causing renal or vesical symptoms.

In most cases Ehrlich's *diazobenzol reaction* is obtainable. The test is performed in the following way : A solution is made consisting of 2 grammes of sulphanilic acid, 50 c.c. hydrochloric acid, and 1,000 c.c. distilled water. A second solution consists of 0·5 per cent. sodium nitrite in water. Immediately before use, 50 parts of the first are mixed with 1 part of the second solution (fresh diazosulphobenzol being thus obtained). Equal parts of this mixture and of the urine to be examined are mixed in a test-tube, after which strong solution of ammonia is added. The appearance of a crimson or rose colour means a positive result. The ammonia may be mixed with the contents of the tube at once, or may in the first place be floated upon them. In the former instance, the entire contents show the colour ; in the latter, it is seen at the plane of contact. The test-tube should thereafter be thoroughly shaken, and the reaction is only to be regarded as strongly positive if the characteristic

colour appears in the froth. This reaction, however, is not pathognomonic, as it is sometimes found in tuberculosis and other febrile diseases.

Peripheral neuritis, the 'typhoid spine' (a painful condition of the back, probably neuralgic), hemiplegia (from arterial obstruction), insanity, neurasthenia, otorrhœa (especially in children), cholecystitis, gallstones, pyelitis, cystitis, orchitis, parotitis, arthritis, and periostitis (with or without necrosis), are occasional sequels that may be mentioned. When abscesses occur in the periosteum or elsewhere, they may be due to the typhoid bacillus alone, but some more common pyogenic microbe may be present. Septicæmia may result from a general secondary infection—for instance, by the *Staphylococcus pyogenes aureus*. Pregnant women often miscarry.

Diagnosis.—In many instances this cannot be made with certainty during the first week, though the gradual onset of ill-health with abdominal disturbance and pyrexia should suggest it from the first. In this country, when fever continues for a week without a characteristic rash, and without local inflammation to account for it, either enteric or tuberculosis should be suspected.

In *tuberculosis* there is frequently evidence that there is special involvement of particular organs, such as the lungs (cyanosis, very rapid breathing, moist râles all over the lungs, and possibly an 'inverse temperature'—the morning record being higher than that of the evening), or the meninges (vomiting, retraction of the abdomen, constipation, infrequency of the pulse, irritation or palsy of cranial nerves). If the localising symptoms are abdominal, there is likely to be a longer duration of the illness, and a more irregular temperature-curve than in enteric. If for a time there is a very irregular or an inverse temperature, with no localising phenomena, the discovery of tubercles in the choroid, or a history of some preceding tubercular lesion, may point to tuberculosis.

Typhus is distinguished by the bloated look, the congested conjunctivæ, the sudden onset, the absence of abdominal symptoms, the petechial rash, which is often seen on the

backs of the hands and feet, and the termination by crisis.

Lobar pneumonia, when not associated with enteric, is distinguished after a few days by the physical signs in the chest. Moreover, the onset is sudden, and the respiration and pulse are greatly accelerated. Herpes labialis, common in pneumonia, is rare in enteric. In enteric there is no leucocytosis, whereas in pneumonia, unless the attack is either very trifling or of intense severity, leucocytosis is well marked.

Influenza sometimes suggests enteric, but its onset is more sudden and its course much shorter. Moreover, the pains in the head, back, and legs are often almost characteristic.

Other diseases which ought to be borne in mind in doubtful cases are ulcerative endocarditis, pyæmia, malarial fever, simple gastro-intestinal catarrh, and appendicitis. The early erythematous blush in some cases of enteric should not lead to a mistaken diagnosis of scarlet fever.

There remains to be mentioned one of the most important clinical tests of enteric fever, viz., *Widal's reaction*. A strongly positive Widal's reaction may be taken as important, though not conclusive, evidence that the individual is suffering now, or has suffered in the past, from infection by the *Bacillus typhosus*. A negative result is not of very great value. Cases which give no reaction may yet manifest characteristic symptoms, prove fatal, and allow of the diagnosis based on the presence of the usual symptoms being confirmed by the autopsy. Moreover, the reaction does not usually appear till about the end of the first week of the disease ; it may be delayed much longer, and, after appearing temporarily, it may vanish. After the attack is past, it may remain for only a few days, or may persist indefinitely. It has been found very marked more than twenty years after the illness.

A positive Widal's reaction may be obtained in cases where the enteric bacillus has attacked, not the intestine, but some other organ, such as the spleen.[1] It is also

[1] Monro and Campbell, *Trans. Glasg. Path. and Clin. Soc.*, May, 1904 ; Eve and Thresh, *Med. Press and Circ.*, May, 1904.

frequently observed apart from the enteric infection altogether, as in general tuberculosis.

To carry out the test, a drop of blood is obtained from the patient and diluted with 50 or 100 times its bulk of sterile bouillon. The blood may be transferred on a coverslip or slide, but if it has to be sent to a distance, or if the test cannot be carried out promptly, it should be collected with aseptic precautions in a capillary-tube, which may then be sealed at the ends. Of this diluted blood, a hanging drop is made, and the hanging drop is then inoculated with a culture of typhoid bacilli (agar, bouillon, or any other). If the blood be from an enteric case, the bacilli, at first freely motile and scattered over the field, become motionless and collect into masses (' agglutination ' or ' clumping '). Under ordinary circumstances, with a dilution of 1 to 50, the reaction will be obvious in half an hour, but the observation must be prolonged for two hours before the result can confidently be recorded as negative. If serum from a blister be employed instead of blood, a dilution of 20 or 30 is sufficient.

Different writers recommend different methods of carrying out the test, and some consider it very important that the culture of typhoid bacilli should be young, the organisms being then particularly motile.

The theory of the test is that in enteric fever an antisubstance (viz., an agglutinin) is developed in the blood which has the power of influencing typhoid bacilli in the way indicated. This substance is probably different from the antisubstance (viz., the immune-body), which, acting in co-operation with the complement, confers immunity on the patient.

Prognosis.—Epidemics vary greatly in severity, but the mortality in this country may be reckoned at from 10 to 20 per cent. In childhood the outlook is better than in adult life. Alcoholism, pregnancy, and delay in taking to bed tell against the patient. High fever with almost no morning remission, a pulse as high as 120 in an adult, incontinence of urine and fæces, insomnia, great abdominal distension, and the typhoid state, are of evil omen. Complications naturally

add to the gravity of the case, though in very varying degree. Hæmorrhage is sometimes, and perforation almost always, fatal.

Enteric fever generally confers immunity against a second attack, but not so constantly as do some other infections.

Treatment.—The sooner the patient takes to bed the better. Careful nursing is important, and different beds for day and night are an advantage. The evacuations ought to be passed into a bed-pan, but some male patients have great difficulty with this, and it is better to allow such to sit on a night-stool beside the bed. The patient should be put on a fever diet—frequent small meals of liquid food (diluted milk, whey, beef-tea, barley-water, soups without vegetables, white of egg and water, and an abundance of plain water). Nothing must be given which could irritate the intestinal lesion or, by causing local spasm of the intestine, bring about perforation. In severe cases with feeble heart, rapid pulse, and grave nervous symptoms, stimulants should be given in the form of strychnine (hypodermically), ether, and ammonia. Alcohol may be tried as a sedative, and quinine may also be given.

Constipation should be relieved by enemata. Purgatives, at least after the first few days, must be altogether avoided. If the motions exceed two or three daily, the milk may be boiled and mixed with lime-water. More severe diarrhœa may be treated by enemata of starch and laudanum, or by a mixture containing acetate of lead and opium. Severe abdominal distension may be treated by salol or salicylate of bismuth, by pepsin, or by fomentations. For insomnia, opium may be used, and for troublesome delirium, icebags should be applied to the head.

If hæmorrhage occurs, opium should be given frequently, and the quantity of food should be reduced for a few days. Ice may be allowed to dissolve in the mouth and rectum. Acetate of lead may be given with the opium ; or turpentine may be given in 20-minim doses every two hours ; or, again, calcium chloride (5 grains every four hours) may be administered with the milk, whilst ergotin is given hypodermically.

When perforation occurs, the patient's miserable chance

is greatly improved if laparotomy is at once undertaken, and by this means about a fourth of the cases can be saved. The case is obviously very different from that of a ruptured gastric ulcer, since in enteric not only is the general condition very low owing to the long-continued fever, but there are probably quite a number of intestinal ulcers ready to perforate whenever the bowel is manipulated. Failing operation, the treatment of perforation is by opium in large doses, not only to relieve pain, but also to quiet the movements of the intestine until adhesions take place.

Thrombosis of the femoral vein is to be treated by continued rest and by the application of glycerin and belladonna to the seat of pain. Friction must be avoided, lest a portion of the clot be detached, and give rise to embolism of the pulmonary artery.

During convalescence the utmost care must be taken with the diet. No solid food should be given until the evening temperature has been normal for five days or a week. The heavier articles of diet—vegetables, fruit, red flesh, etc.—should not be given till about a fortnight later. Perforation is the greatest danger to be thus guarded against. After a severe attack the patient should not return to work for several months. He should live in the country, and have every possible advantage as regards food, clothing, rest, and fresh air.

It remains to be mentioned that various procedures have been recommended for the routine treatment of typhoid fever apart altogether from the question of complications. One of these is the *antipyretic* method, which aims at preventing the temperature from going above 102·5° F. If this is exceeded the patient is put into a cool bath (70° to 85° F.) for a quarter of an hour, during which time he is rubbed so as to promote the peripheral circulation. The bath is repeated every three hours as long as the temperature tends to exceed the height stated. Cold spraying, the cold pack, and cold compresses may be employed with a similar end in view. The evidence is conclusive that the cold-bath treatment, when strictly carried out in hospitals, has reduced the mortality of the disease to about 7·5 per cent., as against

12 to 18 per cent. in the absence of hydrotherapeutics. It appears to do good by promoting elimination even more than by lowering the temperature. Moreover, it favours sleep, and acts as a tonic to the circulatory and nervous systems. Hæmorrhage, peritonitis, phlebitis, and great prostration are contra-indications to the cold bath.

Antipyretic drugs have also been used, but, with the doubtful exception of quinine, their routine administration is objectionable.

Another method is the *antiseptic*. Antiseptics are given by the mouth to act upon the intestinal contents. Among the numerous drugs which have been thus used are calomel, salol, and β-naphthol. They are probably harmless as a rule, but they do not shorten the disease.

Vaccination against enteric was introduced by Wright some years ago with encouraging results. The vaccine consists of cultures of typhoid bacilli killed by heat, and two inoculations are given at an interval of about a fortnight. This method of preventive inoculation appears to diminish both the susceptibility to the disease and the case-mortality.

Chantemesse has recently published encouraging results obtained by the use of an antityphoid serum procured from a horse which was rendered immune by injections of soluble typhoid toxin. Unfortunately, however, no such toxin is yet available for general use.

To prevent the spread of the disease, it is important to disinfect the excreta before they are put into the drains. A 5 per cent. solution of carbolic acid with a 4 per cent. solution of chlorinated lime may be mixed with an equal quantity of the dejecta and allowed to stand for an hour. Or perchloride of mercury, 1 in 1,000, kept in solution by hydrochloric acid, 2 per 1,000, and coloured with aniline; or carbolic acid, 1 in 20, by itself, may be employed for the same purpose. There is reason to believe that the urine of a person convalescent from enteric is more liable to spread infection than the fæces, and for some weeks after recovery the urine should still be disinfected. This can be done by the regular internal administration of urotropin (a combina-

tion of formaldehyde and ammonia), which seldom causes any inconvenience. Five or ten grains may be given thrice daily.

Paratyphoid Fever.—This infection is due to the paratyphoid bacillus, an organism whose characters place it in an intermediate position between the *Bacillus typhosus* and the *Bacillus coli communis*. The clinical features of paratyphoid fever are almost identical with those of enteric, including a gradual onset, splenic enlargement, frequently rose-spots and diarrhœa, and occasionally intestinal hæmorrhage. The disease may be mild or severe, and may relapse. Epidemics have been traced to the drinking of infected water.

The disease differs from enteric fever in possessing no characteristic morbid anatomy. Peyer's patches, the solitary follicles, and the mesenteric glands remain healthy. Though the intestine is in some cases extensively ulcerated, the lesions resemble those of dysentery rather than those of enteric. The spleen is always enlarged.

The diagnosis of paratyphoid fever is justified when Widal's reaction is either negative, or positive only in very low dilution ; when the paratyphoid bacillus can be cultivated from the patient's blood, urine, or stools ; and when both this culture and a known paratyphoid culture are agglutinated by the patient's serum.

The prognosis is favourable, since very few recorded cases have proved fatal. The treatment is similar to that of enteric fever.

3. RELAPSING FEVER

(Famine Fever. Spirillum Fever. Seven-Day Fever. Febris Recurrens).

Definition.—An acute infectious fever, characterised by the presence of a specific spirillum in the blood, by a sudden onset and crisis, and by a relapse which sets in about a week after the crisis.

Etiology.—The *immediate cause* of the disease is the *Spirochæta Obermeieri*, an organism which shows about ten

spirals and has pointed ends.[1] Its length is 10 to 40 μ, or several times the diameter of a red corpuscle. During the febrile period it is present in the blood, where it is actively motile. It disappears at the crisis and reappears at the relapse. It is believed that in the interval the organisms which caused the original attack are destroyed by cells in the spleen. At this period small glistening bodies, supposed to be spores, can be seen in the blood. The spirochæte can be kept alive in blood outside the body for a fortnight, but has not yet been artificially cultivated. It is easily stained in dried blood films. The disease can be inoculated in human beings and in monkeys by means of the blood of patients. It is endemic in India, but has occurred as an epidemic in Ireland and other temperate regions.

Of the *predisposing causes*, destitution, including want of food, want of fresh air, and want of cleanliness, is the most important. Hence epidemics of the disease have been specially associated with famines. Overcrowding favours its spread, which takes place from patient to patient through the air, or sometimes by the medium of fomites. The spirochætes are not found in the secretions or excretions, and are probably transmitted by the common bed-bug. Dutton and Todd concluded that the *tick fever* of the Congo Free State is relapsing fever due to a spirillum which is probably identical with the *Spirochæta Obermeieri*, and that this organism can be transmitted by the bite of the *Ornithodoros moubata*, or horse tick, an animal whose habits closely resemble those of the bed-bug. An attack of tick fever confers immunity. Males suffer from relapsing fever more than females. The age between puberty and maturity is specially susceptible.

Morbid Anatomy.—The changes found after death are not

[1] The tendency of the present time is to regard the spirochætes of blood as protozoa rather than bacteria. They do not grow on ordinary culture media; the course of the fever they induce is unlike that associated with bacterial infections; the spirillar disease of fowls is conveyed by ticks, and in this respect resembles various other protozoan infections; and, finally, Schaudinn's researches on the life-history of *S. Ziemanni* make the evidence for their protozoan nature very strong.

characteristic. The spleen and liver are enlarged. In addition to the usual results of fever, there may be necrotic areas in the spleen containing numerous spirochætes.

Incubation.—The incubation period varies greatly. It may be a few hours, or it may extend to three weeks.

Symptoms.—The patient is suddenly seized with severe headache and shivering, and the temperature rises fast and high (104° to 109°). Other early symptoms are giddiness, thirst, anorexia, sickness, and pains in the back and limbs. The pains are both muscular and articular. The tongue is foul, the skin is dry and jaundiced, and the liver and spleen are enlarged and tender. There is usually constipation, but in some cases there is diarrhœa of a dysenteric type. In exceptional cases there is an eruption of rose-spots, which are smaller than those of enteric fever.

After a period of from five to seven days, defervescence takes place with great rapidity, and as the temperature generally falls to several degrees below the normal, the crisis may represent a difference of more than 10° F. within the twenty-four hours. The crisis is associated with profuse sweating, diarrhœa, and diuresis, and sometimes with hæmorrhage from mucous surfaces. The temperature thereafter gradually rises to the normal, and after a few days the patient feels quite well, and, if in hospital, is probably anxious to get home. But a relapse suddenly occurs about a week after the crisis. This is practically a repetition of the original attack, except that it is about two days shorter. Convalescence, which is tedious, usually dates from the second crisis ; but there may be several relapses, or in rare instances none at all.

Complications.—Among these may be mentioned cardiac failure, bronchitis, pneumonia, diarrhœa, dysentery, and hæmorrhages. Pregnant women always abort, usually in course of the relapse. Premature labour commonly involves death to the fœtus and is dangerous to the mother, one of the risks being from post-partum hæmorrhage. Ophthalmia sometimes supervenes in early convalescence.

Diagnosis.—The symptoms in general, the crisis after one week, and the relapse a week later, are sufficiently character-

istic. Or the blood may be examined, either in the fresh state, or after staining of a film with some aniline colour.

Apart from the relapse, and the results of examination of the blood, the disease may be distinguished from *typhus* by the higher initial temperature, the jaundice, the severe pains in the back, and the absence of the specific eruption, bloated appearance, and marked delirium of typhus. The presence of an epidemic of the disease is, of course, an important guide towards the diagnosis.

Small-pox is soon recognisable by the eruption.

Rheumatic fever is associated with swelling of the joints, and the muscles are little, if at all, affected.

Enteric fever is characterised by a gradual onset, diarrhœa, and other abdominal symptoms.

Prognosis.—The disease causes much suffering, but is not very dangerous to life. Death may take place at the height of the original attack, or from collapse after the crisis, or from a complication. The mortality is about 4 per cent., and is chiefly in persons over thirty years of age.

One attack affords little or no protection against a subsequent seizure.

Treatment.—No specific remedy is known. Rest, careful feeding, and good nursing are important. Quinine may be given in the early stages. High fever and severe headache may be relieved by sponging, the cold pack, or applications of ice to the head. Fomentations may be useful if the spleen and liver are very tender. Morphine is indicated for severe pains. In a few cases cardiac stimulants may be appropriate.

4. SMALL-POX (Variola).

Definition.—A specific infectious fever, characterised by a sudden onset, with vomiting, and severe pain in the head and back, and by an eruption which passes through the stages of papule, vesicle, and pustule.

Etiology.—In 1892 Guarnieri described under the name of *Cytoryctes variolæ* certain bodies which he found in the epithelial cells of the skin in small-pox pustules and in vaccinia,

and in the epithelial cells of the cornea in rabbits inoculated with variola or vaccinia. He regarded these 'vaccine bodies' as intracellular parasites, and later researches lend great support to this theory. The supposed parasite is found in the specific lesions of the skin and mucous membranes in small-pox. In early and extending lesions it is present in the protoplasm of the epithelial cell, while in older lesions it is found in the nucleus. If this body is really a parasite, and not simply due to degenerative changes in the cell, it probably belongs to the class sporozoa of the protozoa.

The disease is transmitted by direct contact and by fomites. Opinions differ on the question whether the transmissibility is greatest at the time when the vesicles are developing into pustules, or at the time when the crusts are drying and separating.[1] The virus is given off by the lungs as well as by the skin, and is present in the secretions and evacuations. The infection may travel for a considerable distance by the air. Small-pox prevails mostly in winter and spring. Its incidence depends to an enormous extent on the degree to which a population is protected by vaccination. The average annual death-rate from small-pox in England and Wales has been estimated at one-seventieth of what it was before vaccination was introduced. In former times it was a disease of childhood, because almost every child took it, and those who survived were protected; but in vaccinated communities, where the disease

[1] Before vaccination was generally known, matter from a small-pox vesicle was often inoculated into the skin. The Chinese method was to put small-pox crusts into the nose. Cutaneous inoculation was followed in some cases merely by a local lesion not unlike that of vaccinia, but more frequently by a general eruption. The disease was mild (the mortality has been estimated at 1 in 300, but was not always quite so low), so that immunity was cheaply acquired by the individual in days when almost everyone took small-pox. Unfortunately, however, it was no less contagious than ordinary small-pox, and several epidemics on the Continent of Europe appear to have been started in this way. Inoculation was introduced into England in 1721 by Lady Mary Wortley Montagu, wife of the British Ambassador to Turkey. It was rendered illegal by the Vaccination Act of 1840.

is infrequent, and people are seldom exposed, all ages are liable. The fœtus may be attacked, and may present vesicles or scars at birth.

Morbid Anatomy.—The development of the papule is due to changes in the Malpighian layer (*rete mucosum*) of the epidermis. Some of the epithelial cells become swollen, and by their pressure cause thinning of neighbouring cells, which are thus converted into the septa which divide the cavity of the multilocular vesicle. The swelling of the epithelium being more marked at the periphery than at the centre, the vesicle is umbilicated, but occasionally this phenomenon is attributable to the presence of a hair follicle in the centre of the pock. Hyperæmia of the underlying cutis sets in and leucocytes crowd into the vesicle, converting it into a pustule. The pustule dries into a crust or scab, and fresh epidermis grows underneath it. If the suppurative process involves the true skin, a 'pit' will be produced. The eruption may involve the mucous membranes.

The spleen is enlarged, and cloudy swelling or fatty degeneration may be observed in other viscera. In hæmorrhagic small-pox, extravasations may be found in the viscera and in the mucous and serous membranes.

Incubation.—The incubation period is about twelve days (from nine to fifteen).

Symptoms.—The invasion is abrupt, with shivering, fever, sickness, headache, and intense pain in the lumbar region. In children, convulsions may take the place of shivering; and in severe cases at any age, delirium or coma may set in. The temperature may reach or exceed 104° within a day. The pulse is rapid.

The eruption appears on the third day, but the papules may be felt like shot in the skin before they are actually seen. The characteristic eruption is preceded in some cases by a measly, scarlatiniform, or petechial rash. The erythematous (scarlatiniform, erysipelatous, or measly) *prodromal rashes* may involve the whole surface, or may be localised, and in the latter case are specially apt to affect the extensor aspects of the arms and legs. The petechial rash almost always selects the lower part of the abdomen.

In some instances the initial rash is both erythematous and petechial. These prodromal eruptions generally fade within a few days. Whenever the eruption appears, a great improvement takes place in the general condition, and sometimes the fever quite disappears, so that the patient thinks the attack is at an end. The papules, however, enlarge, and develop at about the sixth day into vesicles which have a flattened pearly appearance. They are depressed in the centre (umbilicated), and are multiiocular. The papules are chiefly found at first about the face and wrists, but they gradually extend to the trunk and limbs, and are often found on the palms and soles, and in the mouth. About the ninth day, the cutaneous lesions suppurate, and their contents become opaque (the vesicles being transformed into pustules); the temperature rises again (*secondary* or *suppurative fever*), and the patient again feels ill. The pustules grow for a time, and become surrounded by a red, inflammatory areola; but about the eleventh or twelfth day desiccation commences, and the pocks dry into crusts, after or without rupturing. After a time these drop off, leaving red elevated spots, which ultimately subside, and give place either to normal appearances or to depressed scars.

The eruption does not confine itself to the skin. It is present on the lips, palate, and other parts of the mouth, and in the pharynx, larynx, gullet, vulva, etc. Dysphagia and salivation may be troublesome symptoms. The eruption may involve the extremity of the urethra and cause great difficulty in micturition. Though conjunctivitis and ulceration of the cornea are common, it is doubtful if pocks actually occur on the eyeball.

In very mild cases, the disease may practically end when the eruption appears, the vesicles scarcely reaching a stage of pustulation. On the other hand, in very severe cases, the improvement in the eruptive stage may be but slight, and a patient who has survived the early stages may succumb to the secondary fever. There may be considerable swelling of the eyelids, face, and scalp. A well-marked leucocytosis is the rule.

Varieties.—In ordinary small-pox, the pocks may remain separate from one another (*discrete*), or may—on the face, hands, and feet, though not on the trunk—be set in close contact with one another (*confluent*). Confluent is more serious than discrete small-pox.

Hæmorrhagic small-pox occurs in two forms. In one type, the usual papular eruption is preceded by an erythematous rash with hæmorrhages in the skin and from mucous membranes (*petechial*, *black*, or *malignant* small-pox, *purpura variolosa*). Death occurs in the first week, sometimes before the characteristic rash appears. In the other type, the disease at first resembles ordinary small-pox, but hæmorrhage takes place into the vesicles or pustules, and often from mucous membranes also. Most cases of this kind die.

Modified small-pox, or *varioloid*, is the form met with in vaccinated persons. As a rule, it is comparatively mild, though the pain in the head and back may be considerable. The eruption is commonly scanty, and may abort at the papular or vesicular stage. There is little or no secondary fever, and scarring rarely takes place.

Complications and Sequels.—These include laryngitis, bronchitis, and broncho-pneumonia ; pleurisy, which is often suppurative ; conjunctivitis, which often leads to keratitis and blindness ; otitis media ; some forms of paralysis ; boils, which are very common ; gangrene of the genitals ; orchitis ; erysipelas ; alopecia ; and scarring (' pitting ') of the skin. Diarrhœa is common in children. Pregnant women usually abort.

Diagnosis.—The pains in the back and head, with vomiting, should at once suggest the disease, particularly if it is prevalent at the time. The prodromal rashes may cause some difficulty at first, but if the absence of coryza and of sore throat is not sufficient for the exclusion of *measles* and *scarlet fever* respectively, the matter is settled in a day or two by the appearance of the papular eruption.

It may be a difficult matter to distinguish between varioloid and *varicella*. In the case of the former, the period of invasion is longer ; the initial symptoms are more severe ; the rash is chiefly on the face and hands (in varicella it is

chiefly on the trunk); and the pocks are hard and shotty, and appear in one crop (in varicella there are superficial blebs, which appear in several crops). The course of the temperature is different in the two diseases. Note should be taken of scars due to a former attack of small-pox or of chicken-pox, and also of vaccination marks.

Syphilis may produce an eruption suggestive of small-pox, but its evolution is very much slower, and the accompanying symptoms will be different.

Prognosis.—The mortality of small-pox in unprotected persons varies in different epidemics from about 25 to 55 per cent., whereas in varioloid the mortality is probably less than 5 per cent. The hæmorrhagic and severe confluent forms are particularly dangerous. Pregnancy influences the prognosis unfavourably. In infancy and old age, the disease is very fatal.

An attack generally protects for life, but this is not invariably the case. Louis XV. died of a second attack.

Treatment.—The patient should be kept in a room at a temperature of 60° F., and should be well supplied with water and other cooling drinks, and with light foods. The pain should be alleviated by opium, and high fever and delirium by the cold bath or pack. The eyes and mouth should be frequently cleaned with antiseptic washes. No satisfactory method is known to prevent pitting, but it is well to cover the face with a mask of lint or cotton-wool. Stimulants may be necessary in severe cases. Tracheotomy is occasionally needed for œdema of the larynx. As soon as the fever subsides, the patient should be allowed to get up, if no complication is present. In convalescence, he should have a daily bath, to get rid of the crusts and scales as thoroughly as possible. As long as crusts are present they ought to be kept moist with vaseline, so as to make them less easily disseminated throughout the room.

If an unprotected person comes under observation within three days after exposure to the contagion of small-pox, he ought to be vaccinated. The period of *quarantine* is sixteen days, beginning with thorough disinfection.

5. VACCINIA (Cow-pox).

Definition.—A specific eruptive disease of bovine animals which, when inoculated into man, gives rise to a local eruption, passing through the stages of papule, vesicle, and pustule, with constitutional disturbance, and which affords more or less permanent protection against small-pox.

Vaccination is the inoculation of the vaccine virus. The symptoms of vaccinia are the same, whether the lymph is obtained from a calf vaccinated for the purpose, or from a human being (*humanised* lymph). Cow-pox in the cow seems to be much less common now than formerly, when it was readily spread by affected milkers, whose hands inoculated the udders. Conversely, healthy milkers who had any abrasions on their hands were apt to be themselves infected (*casual cow-pox*). The general recognition of the value of vaccination is due to Edward Jenner, who in 1796 vaccinated a boy with matter from a dairymaid's hand, and subsequently found that no disease followed inoculation with matter from a small-pox pustule.

Nature of Vaccinia.—There is now sufficient ground for regarding cow-pox as small-pox modified by transmission through the cow. This was Jenner's opinion. In the first place, small-pox and cow-pox protect against one another, and, unless they are the same disease, they are quite peculiar in this respect among the infections.

In the second place, small-pox matter has repeatedly been inoculated in the calf, with the result that cow-pox is induced. Moreover, lymph from such calves gives rise to vaccinia in human beings. It is true that bovines are very refractory to small-pox inoculation, so that the result is often negative, and some have thought this an objection to Jenner's view that cow-pox was originally derived from small-pox in man. But Copeman has found that if human small-pox be inoculated in monkeys, and the contents of the local inoculation vesicles be inserted in the calf, ordinary cow-pox is induced in the calf, and may be transmitted to man therefrom. It may therefore be concluded that the

first cows to suffer from cow-pox suffered in consequence of (accidental) inoculation by the hands of milkers; and that the disease from which these milkers were themselves suffering was the *inoculated* form of small-pox.

In the third place, certain appearances seen in the lesions of small-pox and vaccinia, and probably representing a phase in the evolution of the specific parasite, are common to, and also peculiar to, variola and vaccinia.

It sometimes happens that the inoculation in man of lymph from a calf which has been inoculated with human small-pox gives rise to a general eruption; this may be regarded as small-pox. Moreover, in cases where no such general eruption is induced, the local lesion, even after propagation of the lymph through several calves in succession, may be more severe than when humanised lymph is employed. As cow-pox is a mild or attenuated form of small-pox, it naturally does not confer so permanent a protection as does ordinary small-pox.

Vaccination in Man.—About the third day after the operation, swelling, induration, and redness become observable at the seat of inoculation. The papule grows, and by the fifth day develops into an umbilicated vesicle with milky contents. This vesicle continues to grow, and at about the eighth day begins to be surrounded by an inflammatory areola. This in turn grows in extent until the eleventh or twelfth day, when the local condition begins to subside. At about the end of the second week, the vesicle has dried into a brownish scab; and after another week or so, this falls off, leaving a pitted scar.

Pyrexia and general discomfort accompany the development of the local lesion, and may continue for a week. The lymphatic glands related to the site of inoculation are enlarged and tender, and the movements of the limb are correspondingly embarrassed.

Value of Vaccination.—In a vast majority of cases, successful vaccination renders the individual insusceptible to small-pox for a considerable number of years. The degree of protection, however, diminishes after a time, and children ought to be revaccinated at the age of from seven to

ten years. It is wise to repeat the operation later on in life, and especially if small-pox is prevalent. It is almost certain that universal revaccination would completely abolish small-pox.

The degree of protection conferred by primary vaccination varies directly with the number of marks.

Complications and Sequels.—In rare instances, syphilis and tetanus have been inoculated with vaccine lymph. Erysipelas and abscesses may result through lack of care on the part either of the operator or of the patient. But if proper precautions are taken, the risk of vaccination is reduced almost to zero by the use of glycerinated calf-lymph. The diluted and sterilised glycerin with which the fresh lymph is mixed destroys, before the lymph is used, any microbes that may be present ; so that, after a few weeks, it proves quite sterile when tested by any ordinary culture medium.[1]

Vaccination is commonly performed before the child is six months old, and the usual site is over the insertion of the left deltoid muscle. The skin is thoroughly cleaned, lymph is placed upon it, and cross-scratches are made through the lymph by means of a sterilised needle. It is desirable that there should be four separate marks. The blood should just appear, but should not flow freely from the scratches. The part is then allowed to dry in the air, and a piece of clean linen may be sewn inside the sleeve. In cases where there is any risk of septic infection of the wound, it is better to dress it in accordance with modern surgical principles.

[1] Glycerinated calf-lymph is the vaccine now recognised by Government. If, under any circumstances, humanised lymph has to be used, care must be taken in selecting it. A healthy child, belonging to a family of which the parents and a number of older children are known to the practitioner to be thoroughly healthy, is a good and perfectly safe source of lymph, provided that this can be obtained from well-developed normal vesicles before the areola appears. Small punctures are made on the tops of the vesicles so as not to cause bleeding, and the lymph which exudes is collected on the sterilised needle or lancet for immediate use ; or in capillary tubes, which are thereupon hermetically sealed for transmission.

6. CHICKEN-POX

(Varicella. Water-pox. Glass-pox).

Definition.—An acute specific fever, characterised by an eruption of vesicles on the skin.

Etiology.—The specific virus is not yet known; it is supposed to be transmitted by inhalation. The disease is very infectious, but is met with sporadically as well as in epidemics. Most cases are seen in children of from two to six years of age. It is entirely distinct from small-pox.

Incubation.—The normal incubation period is a fortnight.

Symptoms.—Sometimes the eruption is the first sign of the disease, but it is often preceded by a period of invasion characterised by feverishness and malaise, and occasionally by pain in the back and legs.

The eruption appears within twenty-four hours, and is usually first observed on the trunk or face. Little red papules appear, and in the course of a few hours develop into clear vesicles, whose contents, after a couple of days, become turbid. The vesicles thereafter shrink into brownish scabs, and these separate after a week or two. Several crops of vesicles appear during the first few days, so that the cutaneous lesions can be seen in all stages of development. They are generally few in number, and are always discrete. Sometimes, however, they are present in great abundance, and may then be found in such unusual situations as the palms, soles, and buccal mucous membrane, and may be associated with severe constitutional disturbance. Occasionally the vesicles become umbilicated, and if they are irritated—for instance, by scratching—they are pretty sure to suppurate and leave scars. The temperature in the eruptive period is intermittent or remittent, each rise corresponding to the appearance of a crop of vesicles.

Varieties and Complications.—In rare cases, the vesicles grow so large as to merit the title of bullæ (*Varicella bullosa*). In delicate children, the vesicles may become centres of gangrene (*V. gangrænosa*). In other rare cases, hæmorrhage

takes place into the skin and from mucous surfaces (*V. hæmorrhagica*).

Diagnosis.—The principal difficulty is to distinguish between severe chicken-pox and mild *small-pox* in cases where the eruption is already out when the patient comes under observation. In small-pox, the invasion is marked by severe symptoms ; in chicken-pox, usually by slight symptoms, or by none at all. In small-pox, the whole eruption appears in one crop within two days, after which the temperature falls ; whereas in chicken-pox, there are several crops in the course of several days, each associated with a fresh elevation of temperature. The pocks of varicella are specially abundant on the trunk, those of variola on the face and limbs. Well-marked vaccination scars, or chicken-pox scars, and a history of an earlier attack of either disease, are very important aids in diagnosis.

Prognosis.—Death from chicken-pox is extremely rare.

An attack almost always confers immunity for life.

Treatment.—The principal thing is to prevent the child from scratching. Dusting powders should be applied to the scabs.

The patient should be isolated till all the crusts have disappeared, which commonly means for two or three weeks.

The period of *quarantine* for schools should be twenty days.

7. SCARLET FEVER

(Scarlatina. Febris Rubra).

Definition.—An infectious fever, characterised by sore throat, a diffuse red rash, and a tendency to subsequent inflammation of the middle ear, kidneys, and other parts.

Etiology.—The specific microbe is not yet known with certainty, though Mallory has recently described under the name of *Cyclaster scarlatinalis* a protozoon which he found between the epithelial cells of the epidermis. This lowly animal parasite has also been found in the serum of blisters on the skin of scarlet fever patients. Various other

organisms have been found in cases of this disease, and in particular a streptococcus which is no doubt the cause of the septic phenomena. The infection spreads from person to person directly, or through the medium of fomites or of milk, but not, so far as is known, by water. The virus may be conveyed over long distances and lie dormant for long periods of time. The patient is believed to be most infectious during the height and decline of the eruption. The disease is endemic in large towns, but breaks out from time to time in epidemic form. It is most prevalent in autumn and winter. The great majority of cases occur in the first ten years of life, and the disease is relatively rare after puberty.

Morbid Anatomy.—The appearances are not characteristic, and are largely due to the fever. The spleen is often enlarged. Various degrees of inflammatory change may be present in the throat, with or without secondary changes in the corresponding lymphatic glands. Suppuration of these glands is accounted for by the presence of streptococci. The lymphatic tissue of the intestine may be swollen. Glomerulo-nephritis, endocarditis, pericarditis, etc., may be present as complications.

Incubation.—The usual incubation period is two days (from a day to a week).

Symptoms.—The invasion is generally sudden, with shivering, headache, vomiting, pains in the back and limbs, and sore throat. In young children, convulsions frequently occur. The temperature rises high on the first day, the face is flushed, the tongue furred, and the pulse at the outset very rapid. The rash appears on the second day, first on the neck and chest, but soon all over the body. Sometimes there is delirium. There is usually a leucocytosis. The local condition in the throat, the rash, the temperature, and the general symptoms, all increase in intensity for about two days—namely, from the second to the fourth day of the symptoms—and then a gradual improvement sets in all round, defervescence being by lysis. The fever and the rash have commonly disappeared by the end of the week.

The tongue is at first red at the tip and edges, and covered in the centre with a white fur, through which the swollen papillæ project. This is the 'strawberry tongue' recognised by some writers, whereas others apply the designation to the later stage, when the fur has disappeared from all parts of the organ, and the fungiform papillæ remain unduly conspicuous. This later stage is also called the 'raspberry tongue.' The soreness of the throat is associated with redness and swelling of the fauces and tonsils, and the submaxillary glands may be enlarged and tender.

The eruption is a bright-red diffuse flush, in the midst of which are scattered very small red papules. On the face, palms, and soles, however, the rash consists simply of a diffuse red blush without the punctate element. In the case of the face, it is most marked on the cheeks, and it is noteworthy that it remains entirely absent from the skin around the mouth. (This circum-oral pallor, bounded by the naso-labial furrows, is seen also in pneumonia.) As the rash fades, or even earlier, desquamation commences. This varies greatly in amount and character. It may be branny or it may be extensive peeling, so that patients occasionally secure the skin of their hands in the form of gloves. The amount depends largely on the intensity of the preceding eruption. The process generally occupies several weeks, and occasionally some months.

Varieties.—The description just given applies to what is sometimes called the *simple* or *benign* type of scarlet fever. But in some cases the throat is scarcely affected at all, and in some the whole attack is so trivial that it is neglected at the time, possibly to be brought under notice subsequently by the development of anasarca (*latent scarlatina*). Occasionally a mild case, otherwise characteristic, runs its course under observation without any fever.

On the other hand, the *malignant* or *toxic* variety is associated with great intensity of the specific scarlatinal poison. The throat affection may not be severe, but the temperature is very high and the pulse extremely rapid. Head symptoms are marked, and the rash, which is very pronounced, may become petechial (*hæmorrhagic scarlatina*). Death usually

takes place from coma within a few days, occasionally as early as the second day.

In the *septic*, *ulcerative*, or *anginose* form the throat is not simply inflamed, but ulcerated, and corresponding changes occur in the glands of the neck ('scarlatinal bubo'), while pyrexia is prolonged beyond the usual time in a remittent or intermittent form. A number of these cases recover, but in some there is extensive necrosis of tissue in the region of the fauces, with firm infiltration of the soft tissues of the neck ('collar neck'). Death often occurs in the second week, but the case may continue for some time longer, presenting the symptoms of septicæmia and occasionally of pyæmia.

Surgical and *puerperal scarlatinas* are usually to be regarded as rashes due to septic absorption, or to absorption from the intestine after enemata. If the disease is undoubtedly scarlet fever, its association with the wound or puerperium is probably a mere coincidence.

Complications and Sequels.—*Albuminuria* may be present in the febrile period of scarlatina as of other fevers ('acute febrile albuminuria'), and is then of no great importance. In scarlatina, however, in the second, third, or fourth week from the invasion, it sometimes develops in consequence of *nephritis* ('post-febrile albuminuria'), and particularly glomerulo-nephritis. It may be accompanied by anasarca and hæmaturia. The kidney affection is met with in all degrees of severity. In some cases, death takes place from uræmia; in others, the disease becomes chronic; but in most instances recovery is complete.

Otitis media, due to extension of the inflammation in the throat along the Eustachian tubes, is even more common than nephritis. It is almost confined to the first ten years of life. If the resulting otorrhœa is allowed to become chronic, it may ultimately lead to disease of and within the cranium (mastoid disease, sinus-thrombosis, cerebral or cerebellar abscess, or meningitis). Paralysis of the facial nerve, and deafness, may also result from otitis.

Arthritis ('scarlatinal rheumatism') sometimes occurs in the latter part of the first week, during the defervescence.

One or more joints are swollen and painful, and in rare instances a joint suppurates.

Cervical adenitis has been referred to in connection with the septic type of scarlet fever. The condition sometimes becomes chronic.

Other complications occasionally met with are bronchitis, endocarditis, pericarditis, empyema, chorea, hemiplegia, and, in children, incontinence of urine.

Diagnosis.—*Measles* is distinguished by the coryza, the absence of sore throat, and the later appearance and distinctive features of the rash.

In *rötheln* the rash is measly rather than scarlatiniform, the general symptoms are trifling, and the rash is often the first sign.

Septicæmic and puerperal rashes are recognised by the associated conditions; moreover, the vomiting, headache, and sore throat which characterise the onset of scarlet fever are likely to be absent.

In *exfoliative dermatitis* the throat escapes, and the strawberry tongue is not seen.

Drug eruptions are not associated with fever.

The rash that sometimes follows the administration of an *enema* may closely simulate the scarlatinal eruption. In such cases the history is important, sore throat and fever are absent, and the rash is apt after a time to become associated with urticarious swelling of the face. The patient is almost always a female, and her complaint is generally gastric or pelvic.

Diphtheria is recognised by the presence of the characteristic bacillus in the throat.

It is important to remember that a red blush may appear upon the skin at an early stage of *enteric fever* and *small-pox*.

In a later stage, the occurrence of characteristic desquamation, anasarca, or albuminuria, may throw valuable light upon a doubtful case.

Prognosis.—The mortality varies much according to the age and social circumstances of the sufferers. It is greatest among very young children (10 to 25 per cent.), and among

the poor. In London it is about 4 per cent. all over ; in hospital cases, 5·3 per cent. ; between ten and thirty years of age, less than 1·5 per cent. in hospital cases (Caiger). Great severity of the throat condition or of head symptoms, and pre-existing debility from tubercular or renal disease, are grounds for anxiety.

A patient convalescent from scarlatina is liable to be attacked by other infectious fevers. The most important of these, in order of frequency, are (according to Caiger) diphtheria, chicken-pox, measles, and whooping-cough.

In most instances, but not invariably, an attack confers permanent immunity. A relapse is occasionally observed.

Treatment.—As the streptococcus plays an important part in causing the septic symptoms of scarlatina, the disease has been treated by antistreptococcic sera. One which is said to have yielded encouraging results is a ' polyvalent ' serum derived from several different races of streptococci. This mode of treatment, however, has scarcely yet gained an established position.

In a mild case of scarlet fever no treatment is required beyond isolation, careful nursing in a warm bed, a light diet, in which abundance of milk is included, and regulation of the bowels. In cases with high temperature and delirium, the cold pack may be of great service, and, apart from the pack, an ice-cap may be kept on the head. In severe toxic cases, stimulants may be required. Young children sometimes have to be fed by the nose or rectum.

When the throat is seriously involved, it may be swabbed several times a day with some antiseptic preparation (*e.g.*, carbolic lotion, 1 to 2 per cent., or equal parts of solution of perchloride of iron and glycerin), or it may be irrigated with an acid solution of chlorate of potash. Ice may be given to suck, and poultices may be applied to the neck.

The ears should be carefully watched, and if suppuration occurs, a puncture of the membrane, after cocainisation, may do much to preserve the hearing. Arthritis should be treated by fomentations locally, and by salicylate of sodium or Dover's powder internally. Suppuration in the tissues of the neck will require surgical measures.

During desquamation, the skin should be thoroughly anointed with some greasy preparation to prevent dissemination of the scales, and a warm bath should be given from time to time. On account of the susceptible state of the kidneys, every precaution must be taken for several weeks to avoid chilling ; and for the same reason, and during the same period, it is well to avoid butcher's meat as an article of diet. Where any suppurative complication has taken place, a change to the country, or a sea voyage, is to be recommended as an aid to complete recovery.

The patient should be isolated until desquamation has been completed, and all local changes about the throat, nose, and ears have been attended to.

A child which has been exposed to the risk of infection should undergo *quarantine* for ten days.

8. MEASLES (Morbilli[1]).

Definition.—An acute infectious fever, characterised by catarrh of the respiratory tract and a red eruption.

Etiology.—No specific microbe is yet known. The disease is highly infectious, and is readily transmitted before the symptoms are distinctive. It usually spreads from person to person by the breath, nasal secretions, etc., but may be conveyed by fomites or by a third person. It may be regarded as endemic in most civilised countries, and as it is so widespread and so infectious, it is generally acquired in childhood. The susceptibility diminishes with each year from the age of four onwards, but it is found in Glasgow that there is again an increase in the number of the cases between the ages of seventeen and thirty, this being due to the migration to the city of individuals from country districts which have been long free from measles. When it has attacked virgin soil in recent times, as among the Fiji and Faroe Islanders, it has proved a very formidable disease ; the explanation, doubtless, being that when the

[1] Called also by some 'rubeola,' in which case rubella or rötheln is distinguished as 'rubeola notha' (bastard rubeola).

disease has prevailed in a country for countless generations, a certain amount of resisting power is developed and inherited. In Britain it is most common in June and December. It is predisposed to by any existing catarrh of the respiratory system.

Morbid Anatomy.—The post-mortem appearances are not characteristic; they include hyperæmia of the skin and catarrh of the respiratory tract. Death is almost always due to some complication, and accordingly capillary bronchitis, pulmonary collapse and broncho-pneumonia, are very commonly found after death.

Incubation.—The incubation period is ten days (four to fourteen days).

Symptoms.—The invasion is abrupt, and is characterised by shivering, sneezing, redness and watering of the eyes, running of the nose, sickness, headache, a hoarse cough, and sometimes diarrhœa.

The eruption appears on the fourth day, and both it and the temperature reach their maximum about thirty-six hours later. The rash appears early and strongly on the face, which is often swollen; but it soon extends downwards over the trunk and limbs. It takes the form of small, red, slightly elevated spots, which increase in size and run together to form irregular patches. The latter are separated by healthy skin. The rash generally fades by the seventh day, but a brownish staining may persist for some days longer. A very fine *branny* desquamation follows.

The temperature, after attaining its maximum, falls by crisis.

Before the cutaneous eruption appears, there may frequently be detected on the mucous membrane of the cheeks and lips small red spots with bluish-white centres (Koplik's spots). Diagnostic importance has been attached by Filatow, Koplik, and others to these spots, since they may be present from one to three days earlier than the cutaneous lesions.

Varieties.—The rash may appear unusually early or unusually late; or there may be no rash at all. In rare malignant cases (*toxic* type), the typhoid state develops, and

the rash may be hæmorrhagic.[1] In the *pulmonary* type, grave respiratory symptoms, due to capillary bronchitis or broncho-pneumonia, are present from the commencement.

Complications and Sequels.—These are in great measure exaggerations or extensions of lesions which are normal to the disease. Thus there may be severe laryngitis. Catarrh may extend from the medium-sized bronchial tubes to the capillary tubes, with the result that capillary bronchitis, broncho-pneumonia, and pulmonary collapse develop. Lobar pneumonia is a much less common complication than the lobular type. These pulmonary lesions usually appear at the height of the fever, but sometimes appear in convalescence. Intestinal catarrh may give rise to diarrhœa, and in severe cases this may present dysenteric features. Otitis media may occur, and may go on to suppuration and perforation, and lead to disease of the cranium. Phlyctenular ophthalmia and ulcerative keratitis are other complications. In children, gangrenous stomatitis (*cancrum oris* or *noma*) and gangrenous vulvitis (*noma*) may ensue. Tuberculosis is a very important sequel. In rare instances paralysis may occur. Whooping-cough may closely follow the attack of measles.

Diagnosis.—Even without a history of exposure, the symptoms of the invasion are very suggestive, and the rash in typical cases is conclusive. Koplik's spots are important at an early stage.

In *rötheln* the rash, or the tenderness of the cervical glands, is often the first thing noticed, and the pyrexia is slight.

In *scarlet fever* there are vomiting and sore throat, there is no coryza, the rash appears within one day after the invasion, and is diffuse and not patchy. The temperature falls by lysis in scarlet fever, and by crisis in measles.

Small-pox is recognisable by the severe pain in the back, the rigor, the vomiting, the absence of catarrh, and the development on the third day of the shot-like eruption.

Drug eruptions are not associated with fever.

[1] It is doubtful if the 'black measles' of old writers was really measles. It may have been hæmorrhagic small-pox.

Enema rashes may be in part measly, but are apt to assume a scarlatiniform or urticarious aspect. The throat is unaffected, and the temperature is normal. The fact of an enema having been given may suggest the nature of the eruption. These rashes are chiefly seen in gastric and pelvic cases in females.

Prognosis.—Death rarely results from measles itself, but its pulmonary complications render it one of the most fatal diseases of young children. After the fifth year, however, death is very unusual. The mortality varies enormously in different epidemics, according to the proportion of young children attacked, their hygienic conditions, etc.

Second attacks are occasionally observed.

Treatment.—The patient should be kept in bed in a warm but well ventilated room. The diet should be light. Bright light should be excluded on account of the photophobia. If the rash does not come out well, a hot bath may be given. A simple cough mixture, containing ipecacuanha and glycerin or syrup, may be administered. If troublesome diarrhœa is present, restriction of the diet to milk foods may suffice to relieve it; but if this fails, bismuth or lead, with or without an opiate, should be employed.

In convalescence, great care must be taken to avoid a chill. For debilitated children, tonics, cod-liver oil, and malt extract are desirable. A change of air is also advisable. Isolation should be continued for three weeks after the appearance of the rash, and for a longer period if the catarrh has not passed away by that time.

The period of *quarantine* after exposure to the risk of infection should be at least sixteen days.

9. RÖTHELN

(Rubella. German Measles. Rubeola Notha or Rubeola.[1] Epidemic Roseola. Epidemic Rose Rash. Hybrid Measles. Hybrid Scarlet Fever).

Definition.—An acute, specific, infectious disease of mild type, sometimes attended by fever, and characterised by a

[1] See footnote on p. 59.

measly eruption, enlargement of the cervical glands, and catarrh of the fauces.

Etiology.—No specific microbe is known. The disease spreads by contact, and is often seen in adults.

Morbid Anatomy.—Nothing characteristic is known. Moreover, the disease never kills.

Incubation.—The period of incubation varies from one to three weeks.

Symptoms.—The disease presents features of resemblance both to measles and to scarlet fever, and is apt to be mistaken for one or other of these. At the outset the patient feels a little out of sorts, and the rash appears in the course of a few hours. The throat, nose, and conjunctivæ are moderately inflamed, and the posterior cervical glands are swollen and tender. The rash is often, and the glandular affection occasionally, the first sign of illness. Fever is slight or may be absent. The patient may, indeed, feel quite well the whole time.

The rash generally appears first on the face, and spreads thence over the trunk and limbs. It consists of round or oval, slightly elevated spots, which may run together. After two or three days it fades, and a "branny (*furfuraceous*)" desquamation takes place.

Complications.—These are very rare, but serious involvement of the respiratory system has been known to occur.

Diagnosis.—Rubella is distinguished from *scarlet fever* by the slightness of the symptoms, the absence of vomiting, the appearance of the rash on the first day, the spotted and not continuous character of the rash, and the involvement of the posterior cervical glands.

It is distinguished from *measles* by the trivial character or absence of catarrhal symptoms, by the appearance of the rash on the first day, and by the involvement of the posterior cervical glands.

Prognosis.—Recovery is almost invariable.

A second attack is rare.

Treatment.—As a rule no special treatment is required.

The duration of *quarantine* for schools should be twenty days.

Fourth Disease.—According to "Dukes" there exists a disease which, while presenting points of resemblance to scarlet fever, measles, and rötheln, is distinct from all three. In this fourth disease the body becomes covered in the course of a few hours with a diffuse red rash, the face commonly escaping altogether. The eruption is followed by desquamation.

10. INFLUENZA (Epidemic Catarrh, "Grippe").

Definition.—An acute, epidemic, febrile disease, appearing at irregular intervals, and spreading rapidly and widely; due to a specific micro-organism; assuming very varied aspects in different cases, and liable to be associated with manifold complications and sequels, but especially with diseases of the respiratory system.

Etiology.—The specific organism was described by Pfeiffer and others in 1892. It is present in abundance in the nasal and bronchial secretions, and sometimes reaches the blood. It is also found in the lung when pneumonia complicates the disease. It is one of the smallest bacilli known.

Influenza appears to be endemic in Northern Central Asia, and it travels westward from this region by the principal trade-routes. It has been observed to spread at a rate corresponding to the most rapid mode of communication, such as express trains. It spreads rapidly from person to person, and with the exception of dengue, there is no epidemic disease which attacks such a large proportion of the population within a short time. The virus is transmitted in the sputum directly and through the agency of fomites. It occurs independently of seasonal influence, but the risk of pulmonary complications renders it more formidable in cold than in warm weather. The disease has been more or less constantly present in this country since the winter of 1889-90, when it reappeared after being absent for almost forty years.

Morbid Anatomy.—With the doubtful exception of catarrh of the respiratory tract, there is no anatomical change that

can be recognised as constant. The lesions that are so common in fatal cases are those of complications or of pre-existing disease.

Incubation.—The incubation period is usually less than forty-eight hours.

Symptoms.—In the suddenness of its onset influenza is scarcely rivalled even by pneumonia. The invasion is

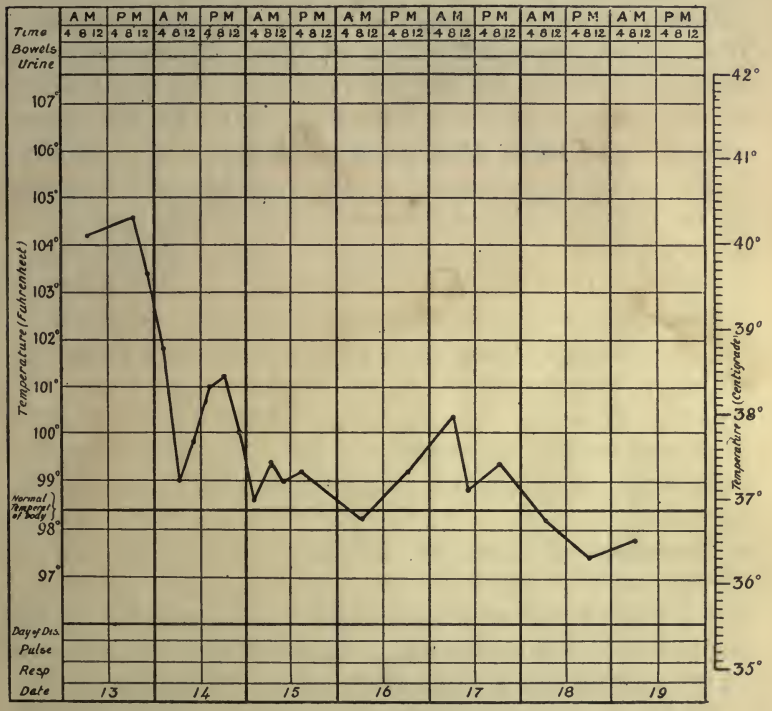

FIG. 9.—INFLUENZA.

characterised by shivering, fever (sometimes exceeding 104° ; Fig. 9), lassitude, and severe aching in the head, back, and legs, and sometimes in the eyeballs. There may be considerable pain in the fauces, with some redness, but without obvious swelling. In some cases, in spite of the severity of the symptoms, no physical signs of local disease can be made out, and localising symptoms may also be absent.

After a few days, if no complication is present, the temperature falls to normal, and the patient is free from symptoms, with the exception of considerable prostration. Very often, however, there is some involvement of the respiratory system. In this *catarrhal* type, the only type which seems to involve the risk that the patient will infect others, there may be a worrying cough without physical signs ; or coryza, bronchitis, pneumonia, or pleurisy. The pneumonia, which is usually lobular, though sometimes lobar, is an important cause of death in epidemics of influenza. In the *nervous* type the pains are particularly severe, and the general prostration great. In the *gastro-intestinal* type there is vomiting, abdominal pain, jaundice, or diarrhœa. In the *febrile* type the pyrexia, which is almost the sole manifestation of the disease, may be very protracted, lasting in rare instances for several weeks. In such cases the spleen may be enlarged. Otitis media is not uncommon, and keratitis is sometimes observed. Herpes is common.

Later Complications and Sequels.—Among these are phthisis, bronchiectasis, and empyema ; endocarditis ; rapidity, slowness, or irregularity of the heart's action ; angina pectoris ; thrombosis of veins ; depression of spirits, melancholia, neurasthenia, insomnia, neuralgia, and multiple neuritis.

Diagnosis.—The epidemic distribution of the disease, the abrupt onset of the fever, the very suggestive pains, and the severe prostration are almost characteristic. If the respiratory tract is affected, a bacteriological examination of the sputum will indicate the nature of the disease.

Prognosis.—Though the case-mortality is very low, the disease attacks such a large proportion of the population that it causes many deaths. It kills chiefly through its pulmonary complications, and by cutting off the subjects of phthisis and heart disease and other debilitated persons.

One attack does not confer immunity against subsequent attacks.

Treatment.—The patient should take to bed at the earliest possible moment, and remain there till the fever has ceased. He ought to be isolated, and the sputum should be dis-

infected. An abundance of light, nourishing food is desirable. Phenacetin (gr. x.) with citrate of caffeine (gr. ii.), or salicylate of sodium (gr. x.) with bromide of potassium (gr. xv.), may be used for the pains, and may be repeated at intervals of a few hours. Phenazone (antipyrin) is specially good for severe pain in the eyeballs. If the heart is feeble, ammonia and strychnine may be required. Respiratory and other complications must be treated as they arise. A change of air and tonics are desirable to complete the convalescence.

II. LOBAR PNEUMONIA

(Acute or Fibrinous or Croupous Pneumonia. Pneumonic or Lung Fever. Peripneumonia. Pneumonia Anginosa. Pneumonitis).

Definition.—An acute infectious fever, due generally, if not always, to Fränkel's pneumococcus, and characterised by local inflammation of one or both lungs and by severe constitutional symptoms.

Etiology.—The specific organism in the majority of cases is the *Diplococcus pneumoniæ* (*Micrococcus lanceolatus*, Fränkel's pneumococcus), which is found in the rusty spit and hepatised lung. It is an oval coccus, and in the sputum is surrounded in pairs by a distinct capsule. This microbe is capable of causing many other forms of inflammation in man (broncho-pneumonia, inflammations of serous membranes, meningitis, endocarditis, otitis media, etc.). Another organism—viz., Friedländer's pneumococcus or pneumobacillus—was formerly regarded as a cause of pneumonia, but recent researches tend to show that acute lobar pneumonia is due to the diplococcus alone. Catarrhal pneumonia and the pneumonia of pyæmic conditions are caused by various micro-organisms.

The diplococcus has been found in the dust of rooms, and is frequently present in the mouths of healthy persons, so that circumstances which diminish the resisting power of the body in general, or of the lungs in particular, are important

factors in the etiology, and we can understand why exposure is so often the apparent cause of an attack. As, however, the organism is often to be found in the blood of pneumonic cases during life, it is quite probable that the microbe reaches the lung in many instances by the blood rather than by the respiratory tract.

The disease is specially prevalent in the late winter and early spring. It occasionally appears in epidemic form. When favoured by defective sanitary conditions, it may haunt particular dwellings ; and in rare instances it is transmitted from person to person, either directly or by the medium of fomites.

Males suffer more frequently than females. One attack predisposes to another, so that a person may suffer from the disease over and over again. Alcoholism and various specific fevers (especially influenza) predispose to pneumonia. An injury to the lung resulting from a blow on the chest is an occasional cause (' contusion pneumonia '). Old people and those who are debilitated by any chronic disease may be carried off by pneumonia. There is reason to fear that the mortality of pneumonia has greatly increased in Scotland during the past half-century.[1] Statistics point to an increase also in the large cities of the United States. Indeed, pneumonia is taking the place of phthisis as the most important cause of death among us.

Morbid Anatomy.—The pneumococcus is found in the affected lung, but in a large proportion of cases it is also present in the blood, and it may thus reach the various organs and give rise to metastatic inflammations. The changes in the lungs are due to a fibrinous inflammation of the alveoli. The process consists of four stages.

In the first stage (*engorgement*, *congestion*, or *splenisation*), the pulmonary capillaries are injected, and serous fluid is present in the air-vesicles. The lung-tissue is dark red, and pits on pressure, though it still floats in water. The alveoli contain leucocytes and red corpuscles, as well as serous fluid and air.

[1] See statistical paper by Alex. Fraser in *Glasgow Medical Journal*, 1902, lvii., pp. 172-179.

In the second stage (*red hepatisation*), the infundibula and vesicles are filled with fibrin and leucocytes, with some red corpuscles. The lung is red, firm, and heavy, sinking in water. Such solid lung accurately conducts to the ear placed over it the respiratory and other sounds produced at the larynx. The red corpuscles in the alveoli give the rusty colour to the sputum in the first and second stages.

In the third stage (*gray hepatisation*), leucocytes are crowded into the alveoli, and by compressing the capillaries cause anæmia of the tissue, so that the lung is pale. Fibrin and red corpuscles are no longer recognisable.

In the fourth stage (*resolution*), the condition reverts to the normal. The cells and fibrin become fatty and soften, and are in great measure absorbed, though sometimes partly removed by expectoration. Sometimes, instead of this, leucocytes continue to accumulate, and they and the fibrin continue to soften till the lung-tissue seems bathed in pus (*purulent infiltration*). It is doubtful how far recovery is possible under these circumstances. In either case, the lung, though still sinking in water, is much less firm, and is slippery, dirty gray in colour, and very easily torn. It is sometimes possible to see the different stages in different parts of a lung. In fatal cases, death very frequently occurs at the period of transition from red to gray hepatisation.

The extent of the disease varies greatly. It often involves one lobe of a lung, or rather more or less than one lobe ; it may involve almost the whole of one lung ; or both lungs may be affected.

The pleura over the inflamed area is almost always coated with a layer of fibrin, and as this may give rise to friction-sound during life, it is possible that the expression ' pleuro-pneumonia ' (as distinct from pneumonia) is scarcely warranted so often as was formerly supposed.

The spleen is frequently enlarged, but by no means always.

Incubation.—The incubation period varies from two days downwards. In one case it appeared to be between twelve and twenty-four hours, and in another it was less than four hours.

Symptoms.—As the disease is met with in a vigorous adult,

the invasion is often very characteristic. In the midst of apparent health, or after being out of sorts, or having an ordinary cold for one or for several days, the patient is seized with a rigor ; and about the same time a severe pain (due to involvement of the pleura) develops in the side of the chest. In some instances the pain is referred to the abdomen, and this has occasionally led to an operation for abdominal disease. In children, convulsions are common at the outset. The temperature rises rapidly. In a day or two the appearances have become so characteristic in typical cases that the diagnosis may be made without touching the patient. He lies on his back with cheeks flushed, breathing very rapidly, but not with difficulty ; there is often an eruption of herpes about the mouth or nose ; there is a short, painful cough ; the sputum is scanty, tenacious, and rust-coloured ; and the chlorides of the urine are deficient or absent. The flush on the face may be specially marked on the same side as the pulmonary lesion. The pulse is much accelerated (100 to 130), but the respiration is proportionately still more disturbed (35 to 60). The temperature is perhaps 103° or 104°, but may be higher. The pyrexia is continuous, though it sometimes varies a good deal in the course of the day (Fig. 2, p. 5). The urine is concentrated.

As the disease progresses the pain becomes less urgent ; the pulse becomes softer ; the tongue is foul, or perhaps dry and nearly clean ; insomnia may be obstinate ; delirium may be present, especially at night ; diarrhœa with green stools is not uncommon, and sometimes the urine is passed into the bed. In drunkards, the delirium may take the form of delirium tremens. Leucocytosis is well marked in favourable cases ; it appears early, and disappears when the pyrexia subsides ; the white corpuscles may number from 15,000 to 40,000 per cubic millimetre.

After a period of from five to ten days, the temperature falls by crisis, usually reaching the normal within twelve hours. The frequency of the pulse and of respiration is greatly lowered at the same time, and there may be profuse sweating (Fig. 10). Sometimes there is a pseudo-crisis a

day or two before the crisis, and occasionally the defervescence is by lysis.

Physical Signs.—The physical signs in the chest do not change nearly so rapidly as the symptoms, either at the invasion or at the crisis. Moreover, they vary greatly in different cases. In a typical attack the decubitus is dorsal,

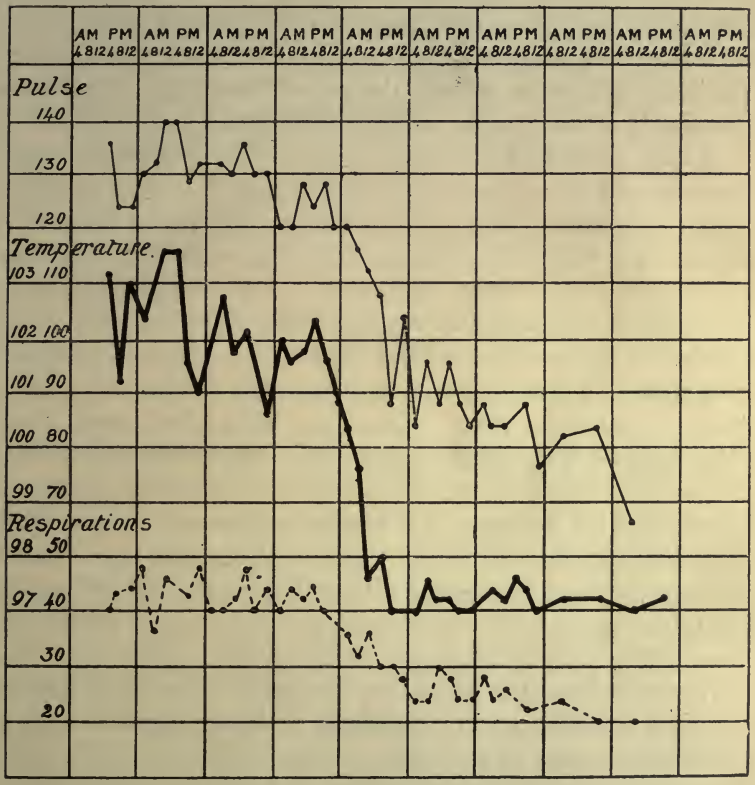

FIG. 10.—PNEUMONIA, WITH RAPID SIMULTANEOUS DECLINE OF TEMPERATURE, PULSE, AND RESPIRATION. (Annie S., æt. 13.)

and the respiratory movements, though rapid, are not embarrassed. Movement is restricted on the affected side of the chest. In the early stage, percussion over the affected portion of lung may be somewhat dull, or it may be tympanitic ('Skodaic resonance'). The respiratory murmur may be much enfeebled, and is frequently accompanied by

a very fine crepitus, which is supposed to be due to separation of the sticky walls of the alveoli, though attributed by some to pleural friction.

After a couple of days, when hepatisation is established, percussion is dull, though often associated with a distinct degree of tympanicity. The respiratory murmur is tubular, and no longer accompanied by crepitus. Vocal fremitus is increased, and whispered pectoriloquy is recognisable. If, as sometimes happens, the bronchial tubes are filled with fibrinous exudation or with thick secretion, there may be no tubular breathing, and vocal fremitus may be diminished.

When resolution sets in, moist râles (*crepitus redux*) become audible. These are coarser than the early crepitations, and result from the bursting of bubbles produced by the air entering the alveoli and mixing with the softening exudation. The other signs are not necessarily altered. The physical signs may be late in developing, and may persist for a considerable time after defervescence. Tympanicity may be present for a long time before dulness is added to it, or takes its place. One part of the lung may yield a tympanitic and another a dull note. Enfeeblement of the respiratory murmur may be far more striking than any change in its quality. In *central* pneumonia, where the consolidated area is deeply seated, there may be almost no abnormal physical signs, but simply the cough, rusty sputum, herpes, and various general symptoms, to indicate the nature of the illness.

As the lesion often spreads in the lung, different groups of physical signs may be recognisable at different parts.

Varieties.—*Latent* pneumonia, where there are the symptoms but not the physical signs of pneumonia, corresponds, no doubt, in most cases, if not always, to a *central* consolidation. In *double* pneumonia, both lungs are involved. It has been supposed that *apex* pneumonia is specially liable to be associated with profound nervous prostration, but the evidence for this is insufficient. *Migratory* or *creeping* pneumonia wanders from one part of the lung-tissue to another, on the same or on the opposite side ; this is one explanation of the gradual defervescence which is occasionally

met with. Pneumonia in old and in alcoholic subjects may set in insidiously. The *terminal* pneumonia of cachectic diseases may manifest itself by almost no symptoms at all. A slight elevation of temperature, and possibly a pain in the side, may lead to an examination of the chest, and in this way a small area of tubular breathing is discovered. Or the patient may be so near death that such an examination cannot be properly carried out. *Secondary* pneumonia follows some other disease, such as influenza, typhus, or diphtheria. *Asthenic* (*adynamic*, *toxic*, or *typhoid*) pneumonia is characterised by intense prostration, the toxæmic phenomena predominating over those referable to the local lesion.

Resolution is sometimes delayed. In such cases, defervescence may be by crisis in the usual way, or by lysis ; but even after many weeks the lung may recover completely.

In rare instances, pneumonia terminates in pulmonary abscess, in gangrene (chiefly in old, alcoholic, diabetic, or otherwise debilitated subjects), in tuberculosis, or, fourthly, in pulmonary cirrhosis.

Complications and Sequels.—Bronchitis may accompany the attack, so that the sputum may be abundant and mucopurulent. The pleurisy may be accompanied by serous as well as fibrinous exudation, or it may go on to suppuration. Endocarditis (benign or ulcerative) and pericarditis (especially in children, and in double or left-sided pneumonia) are not uncommon. Meningitis is occasionally met with, and is attributable, like ulcerative endocarditis, to the pneumococcus ; it may escape recognition during life. Jaundice (probably of the toxæmic variety), membranous colitis, parotitis, otitis media, nephritis, and erythema multiforme are occasional complications. Pneumonia and rheumatism may be associated, and the combination may lead to hyperpyrexia (see Fig. 7, p. 12).

Diagnosis.—In a large proportion of cases, the combination of symptoms and physical signs is unmistakable. In cachectic cases, the risk is of overlooking the pulmonary affection altogether. In acute and chronic alcoholism, the cerebral symptoms may mask all others, and the lungs

should therefore be examined as a matter of routine. In children, the head symptoms may lead to a diagnosis of meningitis. In children, too, pleural effusion may give rise to physical signs very like those of pneumonia, and an exploratory puncture may be required to settle the question. If, however, the dulness is at all extensive, the absence of cardiac displacement is strongly in favour of pneumonia. Acute tuberculo-pneumonic phthisis may be indistinguishable from pneumonia until tubercle bacilli and elastic tissue appear in the sputum. While careful watch must be kept over the condition of the lungs and heart and their serous membranes, it is very important to avoid much shifting of the patient and prolonged examinations. The back of the chest should be reached by rolling the patient on to his side, and not by letting him sit up.

Prognosis.—Different statistics show rates of mortality varying within limits as wide as 6 per cent. and 38 per cent.; about 18 per cent. appears to be the average of all cases. Above twenty years of age, it rises with each decade. Chronic alcoholism, chronic visceral disease, and a recent attack of influenza are unfavourable elements in prognosis. The absence of leucocytosis is very ominous, even though every other feature in the case would justify a favourable outlook. The brown, watery sputum, known as 'prune-juice' expectoration, and pointing to the supervention of pulmonary œdema, is a bad sign. A pulse persistently above 120 in an adult, a hyperdicrotic pulse, and an irregular pulse are unfavourable. Sudden collapse may occur and prove fatal. Complications may add greatly to the gravity of the case. Persistent severe pain or distress in the chest generally, or in the cardiac region in particular, should suggest pericarditis. The toxæmia of the disease bears no constant relation to the extent of lung involved.

Death is rarely due directly to the disease of the lungs, but is often due to cardiac failure, sometimes to toxic poisoning of the respiratory or other nerve-centres, and sometimes to complications.

Pulmonary gangrene and abscess, though dangerous, are not necessarily fatal.

One attack of pneumonia confers no immunity against a later attack. Recurrences are frequent. The late Sir James Paget had six attacks in twenty years,[1] and a case has been recorded in which there were twenty-eight attacks.

Treatment.—As the disease cannot be arrested by any means yet known, the treatment is almost wholly symptomatic. The diet should be that which is adapted to the febrile state, but care should be taken not to give too much liquid, lest flatulent distension of the abdomen should embarrass the heart's action. Bleeding may be performed in robust subjects at the very outset. Antipneumococcic serum (20 c.c. injected subcutaneously twice a day) has been recommended by some, but has not yet been generally accepted as beneficial. For severe pain in the side, morphine may be injected at the commencement; but later on, opiates must only be used with the utmost caution, on account of their influence on the respiratory centre. Poultices, either iced or hot, may also be used to relieve the pain. If the cough is troublesome, a mixture containing spirit of chloroform and carbonate of ammonium is suitable. To allay fever, ice-bags may be applied over the affected side of the chest, and the limbs should be sponged with cold water. Hyperpyrexia must be treated by a cold or tepid bath. Severe head symptoms may be relieved by the bath or by the cold pack. Cardiac stimulants are indicated in many cases, and very specially in presence of marked head symptoms, with a hard, dry tongue and very soft pulse. In recent years there has been a strongly growing tendency to remove alcohol from the list of stimulants of the cardiac muscle. The results of experiments on animals are certainly against it. It may, however, be occasionally of service as a narcotic—for instance, in the insomnia of the typhoid state. Under such circumstances the best plan is to give a few doses experimentally and watch the results. If the pulse-rate comes down, the tongue becomes less dry, and the restlessness abates, alcohol should be continued. A dose of whisky may also be comforting to the patient in the collapse of the crisis. Alcohol is sometimes advocated as a

[1] 'Memoirs and Letters,' p. 190.

food, but in pneumonia there is seldom any serious difficulty in getting the patient to take an abundance of milk. Where alcohol is employed it should be given in the form of very old brandy or whisky, the daily allowance of either being from 3 to 10 ounces. From the beginning of any serious case strychnine should be given hypodermically ($\frac{1}{60}$ to $\frac{1}{30}$ grain every few hours), and ammonia with strophanthus may be administered by the mouth several times a day. Hypodermic injections or enemata of saline solution (1 to 2 pints) may be employed as an attempt to promote the removal of toxin from the body. Oxygen gas, well diluted with air, should be tried if there is much cyanosis. A suitable hypnotic is greatly needed; the safe ones are apt to fail, and the powerful ones are not safe in this disease. Paraldehyde, even in 3-drachm doses, is apt to increase the delirium. In some cases morphine can be given in small quantity for this purpose with good result; and if the patient seems likely to sink from exhaustion, arising largely from his insomnia, it may be justifiable to run some risk to procure sleep.

The sputum of pneumonia ought to be disinfected, and attention should be given to any dwelling that appears to be a haunt of the disease.

12. EPIDEMIC CEREBRO-SPINAL MENINGITIS

(Cerebro-spinal Fever. Cerebral Typhus. Spotted Fever. Petechial Fever. Malignant Purpuric Fever).

Definition.—An infectious fever, occurring in epidemics and sporadically, caused by a specific micro-organism, and characterised by inflammation of the cerebro-spinal membranes and an irregular course.

Etiology.—The organism is the *Diplococcus intracellularis meningitidis*, or *meningococcus*, which was isolated by Weichselbaum in 1887. It occurs mostly within the cells of the exudation. It is a small diplococcus which is not unlike the gonococcus. It is to be noted, however, that

sporadic cases characterised by the same anatomical and clinical features may apparently be due to another organism, such as the pneumococcus, streptococcus, staphylococcus, or *Bacillus typhosus*.

The disease does not pass directly from person to person, nor is it transmitted by the excreta or by fomites. It may occur in the form of a house epidemic, exactly as in the case of pneumonia. It attacks principally children and young adults. Epidemics are seldom widespread, and are of rare occurrence in Britain ; they occur mostly in winter and spring. Sporadic cases, however, are not uncommon in this country. The disease is favoured by defective hygienic conditions and by fatigue. It is probable that the microbe reaches the membranes by way of the nose and naso-pharynx. It has been found in the nasal secretion and in the blood of patients suffering from this disease.

Morbid Anatomy.—In malignant cases, there may be simply intense hyperæmia of the brain and cord. In acute cases, there is a purulent or fibrino-purulent exudation in the soft membranes, distributed irregularly over both base and vertex of the brain, and in the case of the spinal cord most marked on the posterior surface of the lower portion. The exudate consists chiefly of polynuclear leucocytes. The ventricles are distended with turbid fluid. The cranial nerves and spinal nerve-roots are apt to be embedded in the exudation. In more chronic cases the membranes are thickened. The spleen may or may not be enlarged. Pneumonia or broncho-pneumonia is sometimes present.

Symptoms.—The incubation period is unknown. The invasion is usually sudden, with severe occipital headache, shivering, vomiting, and pyrexia, to which pain and stiffness in the muscles of the neck are soon added. There are pain and tenderness along the spine, and the muscles of the back are rigid. The patient becomes restless and often delirious. Herpes appears about the mouth ; and purpura, erythema, or some other eruption may appear elsewhere. The skin is hyperæsthetic, and there may be abnormal sensitiveness to light and sound. The head is retracted, and squint is often present. The patient lies on one side with the legs drawn

up, and Kernig's sign[1] is observable. The pyrexia is irregular and in no way characteristic. Leucocytosis is well marked.

The duration varies from a few hours to many weeks. Defervescence is gradual. Most fatal cases die in the first week. In cases that survive, convalescence is often very slow.

Varieties.—In the *malignant* type (malignant purpuric fever), death may occur in a few hours, with well-marked hæmorrhages in the skin. The *abortive* type enters on the stage of convalescence after one or two days ; the symptoms may be slight or severe. In the *intermittent* variety, the symptoms intermit or remit every day or two. In the *chronic* form described by some writers, recurrences may take place during several months.

Complications and Sequels.—Among these are coryza, pneumonia, pleurisy, pericarditis, parotitis, and arthritis. Headache may persist for a long time after the attack, and in some instances chronic hydrocephalus develops. The optic and other ocular nerves may be inflamed. There is often inflammation of the labyrinth, and sometimes of the middle ear. Persistent deafness is perhaps the most frequent sequel of this disease, and is a common cause of deaf-mutism in countries where it prevails. If the inflammation damages the substance of the brain or cord, aphasia, hemiplegia, and paraplegia may result, but these often pass away after a time.

Diagnosis.—The most important point is the combination of symptoms pointing to an acute febrile disease of sudden onset, with symptoms of inflammation of the membranes of the brain and cord. Moreover, the disease may be epidemic at the time. To these may be added the cutaneous eruptions, Kernig's sign, complications such as deafness and arthritis, and the results of *lumbar puncture*. To carry out the puncture, the patient is put on the right side, with the body bent far forwards and the knees drawn up. If he is conscious, the skin may be frozen, or a general anæsthetic

[1] *I.e.*, the knee cannot be extended (actively or passively) when the hip is flexed to a right angle. The sign is not confined to spinal meningitis, but may occur also in enteric fever.

may be employed. The large hollow needle is inserted at a little distance from the middle line, just below the spinous process, and is driven upwards and inwards through the space between the third and fourth, or between the fourth and fifth, lumbar vertebræ. The fluid, which is reached at the depth of about 1 inch in infants and 2 inches in adults, commonly flows out drop by drop, and may be collected in a sterile tube. Some of it should be at once centrifugalised and examined for the specific microbe by cover-slip preparations.

It should be remembered that pneumonia and enteric fever may be accompanied by symptoms suggestive of meningitis. Herpes, common in cerebro-spinal fever and in pneumonia, is rare in enteric. Pneumonia and enteric run a definite course, whereas cerebro-spinal fever is very irregular in this respect.

Prognosis.—The mortality varies in different epidemics from 20 to 75 per cent. The mortality is much higher in children than in adults. Coma and recurring convulsions are unfavourable signs.

Second attacks have been occasionally observed.

Treatment.—Morphine given hypodermically is generally recognised as a valuable remedy in this disease. In a vigorous subject, wet cups or leeches may be applied to the back of the neck for the relief of pain. Ice-bags should be applied to the head and spine. Early and repeated lumbar puncture has given encouraging results. It tends to prevent increase of pressure in the cerebro-spinal canal, and it is at least harmless in this disease. Temporary benefit has been reported to follow the introduction of an antiseptic into the cerebro-spinal fluid. About 50 c.c. of cerebro-spinal fluid having been withdrawn by lumbar puncture, the same quantity of normal saline solution (0·9 per cent.) is introduced through the needle. This is followed by 10 c.c. of a 1 per cent. solution of lysol, after which the needle is withdrawn. This procedure is repeated every second day till the cerebro-spinal fluid withdrawn is quite clear and limpid. Otherwise the treatment is symptomatic. Every effort should be made to feed the patient well, as the disease may be very protracted.

13. POSTERIOR BASIC MENINGITIS

(Non-Tubercular, or Simple Occlusive Leptomeningitis. Cervical Opisthotonos of Infants).

This is possibly a form of sporadic cerebro-spinal meningitis, though Still has described as its cause a specific diplococcus, which differs very slightly from the *Diplococcus intracellularis*. Its usual mode of access to the posterior fossa of the base is by the naso-pharynx, Eustachian tube, middle ear, and petro-squamosal suture. The disease is not uncommon in the first six months of life, and is almost confined to children under two years of age. It is most common in winter and spring, and appears to be endemic in large towns.

Morbid Anatomy.—In the early stages of this disease, inflammatory exudation is found in the soft membranes at the base, and especially over the fourth ventricle. After some months the exudation is replaced by thickening and adhesions.

Symptoms.—The onset is abrupt, with head retraction, tonic convulsion and vomiting, soon followed by languor and irritability. The retraction of the head is the most characteristic symptom of the disease, and it usually persists for weeks. There is often much crying. Retraction of the abdomen is rare. Blindness is common, but is not accounted for by optic neuritis ; it may be recovered from completely. Vomiting and emaciation are important symptoms. There is moderate fever. Hydrocephalus often results from adhesions between the cerebellum and medulla oblongata closing the openings in the pia mater. A common time for death to occur is from five weeks to three months after the onset. About 50 per cent. of cases die, 16 per cent. recover completely, and the remainder survive in a state of disablement from hydrocephalus, blindness, deafness, etc.

Treatment.—Great attention should be paid to feeding. Mercurial inunctions and the internal administration of potassium iodide (1 to 3 grains every two hours) should be promptly employed. Puncture of the tympanic membrane has sometimes been followed by improvement.

14. DIPHTHERIA (Membranous Croup).

Definition.—An acute infective disease, due to a specific micro-organism ; characterised by a membranous inflammation of the faucial, nasal, or laryngeal mucous membrane, or occasionally of a wound ; and often causing great prostration, with degenerative changes in the heart, kidneys, and peripheral nerves.

Etiology.—The specific microbe is the Klebs-Löffler bacillus, which is a small bacillus with rounded ends. It stains irregularly, and thus has a segmented appearance. It is present in the membranous exudation, but seldom invades the system unless in very severe cases. The symptoms, therefore, are due to the absorption of the specific toxin. The bacillus can survive for several weeks after being dried, and it grows very well in milk. Contaminated milk often spreads the disease, but drinking-water does not appear to do so. The bacillus may easily be ejected from a diphtheritic throat by coughing or sneezing, and it may then reach a healthy person either immediately or after drying.

The disease is most prevalent in the last three months of the year, and is favoured by a damp, exposed situation. It is endemic in many places in Britain, and especially in large towns ; and its frequency has increased in recent years, owing, no doubt, in great measure to the close manner in which susceptible children are associated in schools. Sporadic cases are met with where no source of infection can be traced. The great majority of cases occur in children under ten years of age, and among the predisposing conditions is a recent attack of acute disease, especially if associated with sore throat—*e.g.*, scarlet fever. The debility that may arise from constant inhalation of sewer gases is possibly also a predisposing factor.

In a large proportion of cases of diphtheria, the throat infection is a mixed one, pyogenic organisms, and especially the streptococcus, being present in addition to the diphtheria bacillus. When general infection takes place this may be

due either to the diphtheria bacillus or to the streptococcus. A patient may suffer from diphtheria and scarlet fever simultaneously.

Morbid Anatomy.—The disease generally begins in the fauces, whence it often spreads into the pharynx and larynx, and sometimes into the nares. It may be limited to the nose, or to the larynx and lower air-passages. In a well-marked case, the false membrane consists of a cell-enclosing fibrinous meshwork, with—in the case of the fauces and nares—the necrosed superficial layers of the mucous membrane, so that in those regions the membrane tends to adhere. In the larynx and trachea, on the other hand, though the mucous membrane is inflamed, it does not die, but simply sheds its surface epithelium, and the false membrane does not adhere.

Patches of lobular pneumonia are present in severe croup. The spleen is often enlarged. The kidneys, in severe diphtheria, are found to be in a state of parenchymatous inflammation or of fatty degeneration. The cardiac muscle may be fatty or granular, while the cardiac ventricles are dilated. Patches of acute parenchymatous degeneration are distributed irregularly in the peripheral nerves. Interstitial neuritis is sometimes found in the nerves of the palate.

Incubation.—The incubation period is about two days (thirty-six hours to seven days).

Symptoms.—The invasion may be either rapid or gradual. The early symptoms are like those of tonsillitis with feverishness, but the soreness of the throat may be very trifling at first ; and as children, in particular, may make no complaint about this part, it is a good rule in practice to examine the fauces of any child suffering from general symptoms whose cause is not apparent. The fauces are swollen and red, and soon present patches of false membrane, whitish, it may be, at first, but afterwards yellowish or dirty gray in colour. This may be adherent loosely or rather firmly, and when it is detached by forceps, the underlying surface is apt to bleed. New membrane soon grows over this raw surface. There may be a discharge from the nostrils, and other evidence that the nares are involved. The tongue is foul,

the glands at the angle of the jaw are swollen and tender, and the urine is very often albuminous. There is usually moderate fever, but the temperature is rather irregular, and is sometimes normal in very bad cases.

Varieties are distinguished as *benign* or *malignant*, according to the severity of the toxæmia and constitutional disturbance; or they are distinguished according to the seat of lesion.

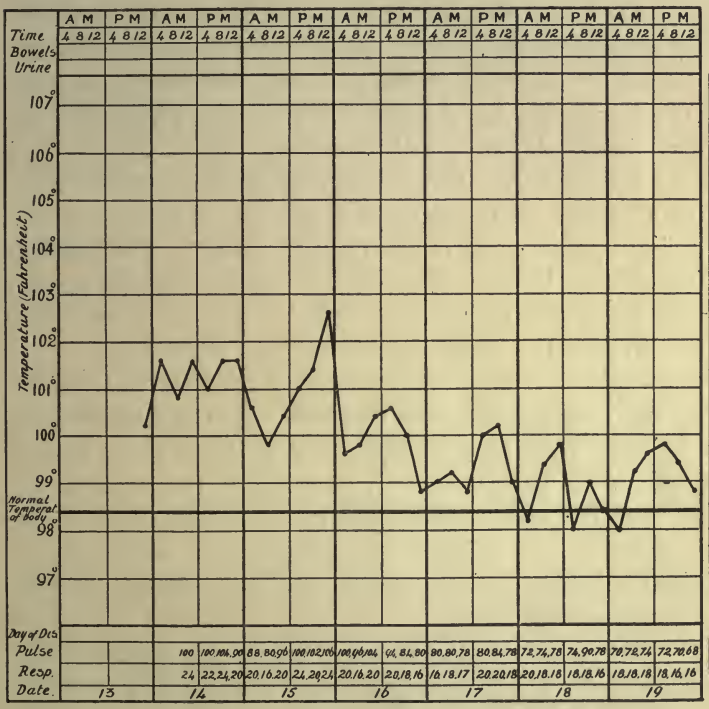

FIG. 11.—DIPHTHERIA IN AN ADULT.

Faucial diphtheria may be either mild or severe. The mild form is seen chiefly in adults (Fig. 11), and sometimes lasts for only a few days. The severe form is seen especially in young children, and is associated with great swelling, an extensive thick membrane, great pain on swallowing, and a tendency to spread to the nares, pharynx, and larynx. In favourable cases, improvement may set in after a week or ten days. In fatal cases, death may occur from toxæmia

and cardiac failure in the second or even in the first week. Even when the local condition is obviously mending, death may occur in the second week or later from gradual or sudden cardiac failure.

Laryngeal diphtheria, or membranous croup, is most common in children, and, though sometimes primary, is generally due to extension from the fauces. The voice is hoarse, and is eventually lost, and breathing is both difficult and noisy. In inspiration, if the obstruction is severe, the larynx is drawn downwards towards the sternum, and the epigastrium, the suprasternal and supraclavicular regions, and the lower ribs are drawn in. The cough acquires the peculiar harsh, ringing quality described as 'croupy,'[1] and dreaded by mothers. The disease tends to spread into the bronchial tubes and pulmonary alveoli, and so to cause increasing cyanosis and death by suffocation. The respiratory difficulty is largely, if not entirely, due to the mechanical obstruction of the larynx and bronchi. In favourable cases, casts of the trachea and bronchi may be coughed up.

Nasal diphtheria may be an extension from the pharynx or fauces, but sometimes occurs by itself. In the latter case it may be very mild, and characterised by little more than a discharge from the nose, with or without pieces of membrane, and by some tenderness of the glands below the jaw. *Fibrinous* or *membranous rhinitis* is a more chronic diphtheria limited to the nares, and not associated with constitutional symptoms.

Membranous inflammation may occur in *wounds*, on the conjunctiva, on the female generative organs, and on other parts, either primarily or secondarily to diphtheria about

[1] 'Croup' is a Scotch word which was in use before the nature of diphtheria was understood. It was applied to a condition of the air-passages associated with a particular variety of cough, and the disease was supposed to be a very severe inflammation. The terms 'croup' and 'croupy,' therefore, have reference to sound. Rokitansky, however, applied the term 'croupous' to pneumonia on account of the fibrinous exudation, in respect of which it had a certain kinship with diphtheria. The word has come to be applied by pathologists to any inflammation associated with a fibrinous exudation.

the throat. It is now known, however, that a membranous sore throat may be due to streptococci or to other organisms quite different from the diphtheria bacillus, and so it is probable that membranous inflammation occurring in wounds, especially when there is no diphtheria elsewhere, is often due to some other microbe than the Klebs-Löffler bacillus.

Complications and Sequels.—A relapse occurs in less than 2 per cent. of cases. Albuminuria is so common as to amount to a symptom rather than a complication; if the patient survives, it almost always passes off. Lobular pneumonia frequently occurs; nephritis very seldom. Endocarditis is a rare complication.[1]

Apart from those just mentioned, the most important complication is *paralysis*, which occurs in about a fifth of the cases which survive the original disease. It is chiefly seen after severe faucial attacks, and sets in most commonly in the second or third week, but may appear much later (in one case after nearly a year). The soft palate suffers most; the loss of power gives a nasal quality to the voice, and allows food to regurgitate through the nose. The next most important palsy is that of the ciliary muscle, which causes loss of accommodation. The third disturbance of this kind, in order of frequency, is in the legs, which suffer from paresis, inco-ordination, numbness, tingling, and loss of the knee-jerks. Occasionally the muscles of the pharynx and larynx are paralysed, and as this may be associated with anæsthesia of the larynx, the condition is obviously very dangerous. In exceptional instances, the muscles of the upper limbs and back and the external ocular muscles suffer. These various paralytic phenomena are attributable to parenchymatous degeneration of the peripheral nerves resulting from the presence of the diphtheria toxin in the blood. In some instances, however, the palsy of the palate is brought about in another way, viz., as a direct local effect of the primary lesion. In most cases, the patient recovers from post-diphtheritic paralysis in the course of a few weeks or months.

[1] See record of a case, with discussion of the literature, by J. W. Findlay, *Glasgow Medical Journal*, 1903, lix., pp. 27-37.

Diagnosis.—The presence of a membranous exudation was, until some years ago, the essential fact in the diagnosis of diphtheria, but it is now recognised that this lesion may be due to the pneumococcus or streptococcus. According to the usage of the present time, the fact which justifies a diagnosis of diphtheria is the presence in the exudation or discharge of the diphtheria bacillus. Albuminuria, laryngeal symptoms, and a nasal discharge favour a diagnosis of diphtheria in cases of suspicious sore throat. Even a sore throat which is followed by paralysis is not always due to the diphtheria bacillus ; it may be due to the streptococcus or pneumococcus.[1]

Prognosis.—The mortality is influenced by several factors, of which treatment is now one of the most important. Foord Caiger states that by the use of antitoxin after 1894 the mortality in the hospitals of the Metropolitan Asylums Board was reduced from over 30 to about 15 per cent. A country practitioner will tell us that whereas he formerly lost a large proportion of his cases, he now seldom or never has a death from this disease. Involvement of the larynx, great extent and persistence of the membranous exudation in the fauces and neighbouring parts, childhood, and especially early childhood, are unfavourable elements in prognosis. Evidence of an actually failing heart or of intense toxæmia is, of course, extremely grave.

The immunity resulting from an attack seldom lasts more than a month. Second attacks are therefore common, but they are not, as a rule, so severe as primary attacks.

Treatment.—With the doubtful exception of mild cases in adults, antitoxic serum (which is derived from the immunised horse) should always be injected as early as possible. The injection should be made under the skin of the trunk or thigh, with strict aseptic precautions. The dose varies according to the severity of the disease, and not according to the age of the patient. In a mild case, seen soon after the commencement, 2,000 units may be sufficient ; but if the attack is severe, or if the larynx is involved, 8,000 units is a more appropriate dose. Unless in mild cases, a half-

[1] See *Glasgow Medical Journal*, 1901, lvi., pp. 274-277.

dose should be given every day after the first, until the membrane is seen to be separating. In many cases the local and general symptoms undergo a marked improvement after the injection. Tracheotomy is less frequently required than formerly, and the course of the disease after tracheotomy is much more favourable than it was before antitoxin was introduced. Though cutaneous rashes and pains in the joints may follow the injection, no serious or permanent harm ensues. In cases where antitoxic serum does no good, even in very large doses, the presumption is that either the treatment is put in force too late, or the symptoms are largely due to infection by septic organisms.

The serum has been given with good effect by the mouth and by the rectum. It is said to be most efficacious when injected into a vein.

Isolation of the patient and disinfection of the discharges are, of course, essential. The throat should be sprayed or swabbed, and in nasal cases the nostrils should be irrigated, with antiseptic solutions (*e.g.*, formalin 1 in 200, or carbolic acid 1 in 100) several times a day. If the larynx is involved, the patient should be kept in an atmosphere of steam, which may be procured by placing a bronchitis kettle beside the bed, with a tent over both. If a medicine is desired for internal use, a mixture should be recommended containing tincture of the perchloride of iron, potassium chlorate, glycerin, spirit of chloroform and water. If suffocation is impending, tracheotomy or intubation must be performed. Cardiac stimulants will often be required, and strychnine may be given hypodermically at short intervals if the heart's action becomes seriously enfeebled. The patient must be kept lying in bed till all reasonable risk of cardiac failure has passed away. The throat should be regularly treated with formol solution (1 in 4) till the bacilli have disappeared, and the patient ought to remain in isolation until this result has been attained.

Post-diphtheritic palsy does not, as a rule, need special treatment. If, however, swallowing is interfered with, the nasal tube or stomach tube may be required for feeding.

Strychnine as a tonic and a change to the country may be recommended in ordinary cases.

When an individual has been exposed to the risk of infection a *quarantine* of twelve days is to be recommended. In addition, and particularly in the case of children, it may be advisable to administer immediately an immunising injection of from 500 to 1,000 units.

15. WHOOPING-COUGH

(Pertussis. Chin-cough).

Definition.—An acute specific disease, characterised by catarrh of the respiratory tract, and by paroxysms of coughing which terminate with a long inspiration which gives rise to the 'whoop.'

Etiology.—The specific microbe is not yet identified with certainty, though various observers have described micro-organisms which might be the cause of the disease, including a small bacillus resembling that of influenza. The virus is communicated from person to person directly, or by fomites which have been contaminated by expectorated mucus.

Any age is liable, but the great majority of cases occur within the first ten years of life. Epidemics prevail mostly in the colder months of the year. Whooping-cough and measles are supposed to predispose to one another. Thus an epidemic of measles is often followed by an epidemic of whooping-cough.

Morbid Anatomy.—There is no anatomical change distinctive of this disease. In fatal cases pulmonary complications are frequently present.

Incubation.—The incubation period seems to vary a good deal in different cases. About ten days may be regarded as the average.

Symptoms.—Three stages of the disease are recognised. (1) The *prodromal*, *premonitory* or *catarrhal stage*, or *invasion*, generally lasts a week or ten days. The disease seems to be most contagious in this stage. It sets in gradually with

moderate fever, cough and other phenomena of an ordinary feverish cold or bronchial catarrh. A few râles may be heard in the chest, but nothing otherwise abnormal. But as time goes on, instead of improving under treatment, the cough becomes worse and more paroxysmal in character.

(2) In the *paroxysmal stage*, which lasts from three or four weeks to as many months, the characteristic 'whoop' is observed. The paroxysm may occur spontaneously, or be excited by emotion or by some external agency. It commences with a series of short coughs or expiratory explosions, which take place in such quick succession that almost no air enters the chest. The face quickly becomes livid and swollen, the veins are distended, and the eyeballs become prominent; but just as the patient seems on the point of suffocation, a deep inspiration takes place, with a crowing or whooping sound, and the appearance quickly improves. After one or after a series of such paroxysms, some mucus may be expectorated. The paroxysm is frequently followed by vomiting, and occasionally by relaxation of the sphincters. In mild cases there may be only a few paroxysms daily, whereas in severe cases several may occur every hour. They are especially troublesome at night, and young children dread them. On examination of the chest, a few dry râles may be audible, and of course during the whoop nothing else can be heard over the lungs; but, on the whole, the lack of change in the physical signs is a noteworthy fact in view of the severity of the symptoms. One or two little ulcers may develop underneath the tip of the tongue as a result of abrasion by the lower incisors or even by the gum. In this stage, the temperature is generally normal. A well-marked lymphocytosis appears early.

(3) In the *stage of defervescence*, the paroxysms become less frequent and less severe, and in the course of two or three weeks the whoop ceases. It is to be noted that for a considerable time after an attack of this disease the whoop is liable to return in the event of the child taking any other ailment associated with cough.

Complications and Sequels.—Hæmorrhage into the conjunctiva, epistaxis, and other hæmorrhages may occur

through the intense venous congestion of the paroxysm. The convulsions sometimes observed may be due to the cerebral hyperæmia. Hemiplegia occurs in a few cases. Interstitial and vesicular emphysema may result from the severe cough. The constant vomiting leads to emaciation. Sometimes there is diarrhœa. More or less bronchitis is almost constant. Capillary bronchitis and broncho-pneumonia, with pulmonary collapse, are common and very fatal complications. Glycosuria is not uncommon. Tubercular infection may take place, especially in the lungs.

Diagnosis.—The whoop, when present, is sufficiently characteristic, but in the prodromal stage the diagnosis may be impossible. The presence of other cases in the neighbourhood, a paroxysmal tendency, the occurrence of vomiting and the exacerbation by night are of some value.

Prognosis.—Apart from complications, the outlook is favourable, but whooping-cough, with its complications, is a very fatal disease. Young, rickety, or otherwise delicate children suffer most.

Except chicken-pox, there is probably no infectious fever which confers so complete an immunity as does whooping-cough. A second attack almost never occurs.

Treatment.—As the disease is serious, and as it is infectious from the commencement of the prodromal stage, it is manifest that a child ought to be isolated as soon as the disease is suspected. If the attack is severe, the patient should be kept in bed. In the catarrhal stage, a simple febrifuge and expectorant mixture may be given—*e.g.*, solution of ammonium acetate, spirit of nitrous ether, syrup of orange and water. Or ipecacuanha wine may be employed. In the paroxysmal stage, feeding may be rendered difficult by the frequent vomiting, and in that case a very light meal should be given whenever a paroxysm is over. No satisfactory drug is known for the paroxysm, but on the whole belladonna and bromide of potassium are perhaps the two most important. The belladonna or atropine should be given in increasing doses till its physiological effects begin to appear. Osler speaks highly of quinine ($\frac{1}{6}$ of a grain for each month of age ; $1\frac{1}{2}$ grains for each year in children under five years ;

the dose to be given thrice daily). Bromoform (1 to 5 minims in syrup), chloral, butyl-chloral, antipyrin, and phenacetin (1 to 3 grains every four or six hours), are among the drugs recommended for internal use. The fauces and larynx may be painted with a 1 or 2 per cent. solution of resorcin. Change of air, tonics, and cod-liver oil will help to complete the convalescence.

A child should not be allowed to return to school for at least five weeks after the commencement of the paroxysmal stage. The period of *quarantine* after exposure to the risk of infection should extend to three weeks.

16. MUMPS

(Epidemic, Specific, or Infectious Parotitis).

Definition.—A specific infectious fever, characterised by inflammation of the parotid gland, and sometimes associated with inflammation of the testes in males and of the external genitals and mammæ in females.

Etiology.—The specific microbe has not been identified with certainty. It is supposed to be conveyed by the breath and saliva, and to reach the glands by the ducts. The disease generally spreads from person to person directly, but occasionally by the medium of fomites. It is most common between five and fifteen, but may occur at any age. Epidemics prevail mostly in the cold and wet months of the year.

Morbid Anatomy.—The inflammation of the parotid gland is much more interstitial than parenchymatous.

Incubation.—The incubation period is from two to three weeks.

Symptoms.—The invasion is characterised by slight febrile symptoms and by pain about the angle of the jaw. In the course of a day or two, there is well-marked swelling of the parotid as it lies on the side of the neck and cheek. The overlying skin is usually pale and shiny. The swelling may be very tender, and there may be difficulty in opening the mouth to take food, as well as pain in swallowing and on

moving the head. The saliva may be increased or diminished. The parotid of the other side may become inflamed at the same time, but more commonly after a few days. Occasionally the other salivary glands are similarly affected. The parotid swelling subsides after a week or ten days. It almost never suppurates.

A relapse of mumps occasionally takes place, and may be repeated.

Varieties.—There may be no pyrexia, or the constitutional symptoms may be very severe. Orchitis may precede parotitis, or may occur without any inflammation of the salivary glands.

Complications and Sequels.—These include orchitis, otitis media, otitis intima, arthritis, meningitis, and hemiplegia.

In a considerable proportion of males who are past the period of puberty, *orchitis* sets in as the parotitis is subsiding, or still later. It is usually one-sided, and is ushered in by a fresh rise of temperature. It lasts for a few days, and in most cases passes on to atrophy. Boys seldom suffer from this complication. In women there is occasionally inflammation of the vulva or mammæ. Epigastric pain and vomiting, which have occurred in a few cases (without glycosuria), have been regarded as due to pancreatitis.

Diagnosis.—It is important to recognise the swollen parotid gland. The fauces should be inspected, so as to exclude any lesion there, which might cause enlargement of neighbouring lymph glands.

Prognosis.—Mumps is fatal only in rare cases, and usually with meningeal symptoms. A second attack is uncommon.

Treatment.—The patient should be kept in bed, and hot anodyne fomentations should be applied over the swollen gland. For delirium, the ice-cap may be used. In the event of orchitis, the scrotum should be supported and fomented. The bowels should be freely opened from the commencement.

17. RHEUMATIC FEVER

(Acute and Subacute Rheumatism).

Definition.—An acute, non-contagious fever, supposed to be due to an infective agent, and characterised by a tendency to inflammation of fibrous tissues, especially in the joints and heart.

Etiology.—Rheumatism is now regarded as an infectious disease, and different observers have described micro-organisms in connection with it, though the evidence that any of these is the specific cause is scarcely yet conclusive. Poynton and Paine and other writers have described a small micrococcus which occurs in the tissues and in cultures in pairs or short chains. It has been found in the blood and in various lesions in rheumatic fever. Intravenous injections in animals are followed by rheumatic phenomena, including polyarthritis, endocarditis, and chorea. This organism has been called the *Micrococcus rheumaticus*. In different media it produces different acids. One of these is formic acid, which has also been obtained from the urine in rheumatism, as well as from the bodies of the bacteria themselves.

Rheumatism has also been looked upon as a mild septic infection, due to such pyogenic organisms as the *Staphylococcus aureus* and the *Streptococcus pyogenes*. According to this theory, it is not a strictly specific affection. The remarkable influence of the salicylates, however, on the course of the attack is strongly against this view.

The infection is supposed to enter the body as a rule by the tonsils, or it may be by the pharynx. The disease is endemic in moist and temperate climates, but prevails from time to time in epidemic form in a manner similar to certain diseases which are known to be due to microbes. Some of the clinical features of rheumatism, including the number and variety of the lesions, and the sweating, suggest a resemblance to pyæmia and tuberculosis. It resembles the continued and the exanthematous fevers in its tendency to

come to an end spontaneously ; enteric in its tendency to relapse ; and pneumonia and erysipelas in its tendency to recur and to be induced by exposure. It has been suggested that the infective agent thrives specially in damp houses.[1]

As exposure to wet or cold is the most common determining cause of an attack, persons whose occupation entails constant exposure are specially liable to suffer. The disease is not so common in summer as at other parts of the year. Heredity is believed to be influential (Cheadle says it is as influential as in gout) ; age and sex undoubtedly are. Until a few years ago, the mischief that rheumatism does in childhood was not sufficiently recognised, because its manifestations are often unobtrusive. Accordingly, rheumatism was regarded as a disease characterised essentially by polyarthritis, fever, and sweating, occurring chiefly in adolescents and young adults, and complicated in a certain proportion of cases by an affection of the heart.

As a result of the labours of certain London physicians (among whom may be named Barlow, Sturges, Cheadle, and Lees), who have paid particular attention to the rheumatism of childhood, it is now known that cardiac disease without arthritis may be the principal manifestation of rheumatism in childhood ; and when this is borne in mind, our views as to the age liability must be modified. According to Lees, the liability commences at two years of age, and increases to its maximum at ten, after which it gradually declines, to become almost extinct at forty ; and this writer describes rheumatism as 'more frequent, more varied, and more virulent' in the child than in the adult. If all ages are taken together, males suffer more than females, probably in connection with their occupations. But below ten, the

[1] It is, perhaps, well to mention two other theories of rheumatism, though they have now only a historical interest : (1) The *metabolic*, or *chemical*, according to which the lesions and symptoms are due to an accumulation in the body of lactic acid or one of its compounds ; and (2) the *nervous*, or *neurotic*, according to which exposure deranges the nerve centres, which thereupon cause trophic lesions in the joints, etc., either directly, or by altering the metabolism and thus permitting lactic acid to accumulate.

two sexes suffer alike ; while between ten and fifteen girls are far more liable than boys.[1]

Morbid Anatomy.—The arthritis is not characteristic. The fluid in the joints is turbid and albuminous, but not purulent.

Hyperpyrexia is not associated with special changes. There may be serious lesions in the heart, including endocarditis, pericarditis, and myocarditis, sometimes with great dilatation. Pneumonia and pleurisy may be present.

Symptoms.—*Arthritis* is the most obtrusive symptom of acute or subacute rheumatism as ordinarily seen in ADOLESCENTS and ADULTS. The onset is usually abrupt, with some chilliness. The pain often begins in the knee, but soon affects several or many joints, especially those of large and middle size, though the small articulations do not always escape. In some cases only one or two joints are involved. An affected joint is painful, swollen, and tender, and the pain is aggravated by movement. The overlying skin is not, as a rule, changed in colour, but may show a slight blush. The inflammation often leaves a joint almost suddenly and appears in another. This quick flitting from joint to joint is a noteworthy feature of the disease.

Profuse sweating is the rule, and sweat eruptions (sudamina and miliaria) are common. The tongue is moist and covered with a white fur. The urine is febrile, and the bowels are constipated. Delirium is unusual. The temperature usually rises quickly at the onset, pursues an irregularly febrile course (101° to 104°), and falls by lysis. The pyrexia is nearly in proportion to the arthritis. Anæmia develops rapidly, and there is a well-marked leucocytosis. When the fever ceases, the pain is often replaced for some days by a subjective feeling of stiffness in the joints. Very seldom is a joint permanently damaged by rheumatic fever.

[1] Girls are from two to three times as liable as boys (1) to articular rheumatism, (2) to chorea, (3) to rheumatic chorea, (4) to heart disease in rheumatic chorea, (5) to heart disease in chorea where there is no history of rheumatism, and (6) to scarlatinal rheumatism (Cheadle, ' Lectures on the Practice of Medicine,' 1900, pp. 295, 310). Beside these data may be placed the long-recognised fact that mitral stenosis is about three times as common in women as in men.

In a certain proportion of these cases—perhaps a third of those that begin with distinct arthritis—*endocarditis* of the simple or benign acute type develops. The tendency to this inflammation increases with the number of attacks of rheumatism, but diminishes as the age of the patient increases. It is signalised by a murmur indicative most commonly of mitral disease, less frequently of aortic valve disease. It is to be noted, however, that systolic murmurs in rheumatism may be due to the blood state (hæmic murmurs), or to relative incompetence of the mitral valve, and not to structural change in the valve. It is often impossible to decide what is the exact state of matters.

Much less frequently *pericarditis* with effusion takes place. This is indicated by friction sound and by an upward extension of the cardiac dulness. If the effusion becomes great, the dulness extends to the right of the sternum. If the effusion is such as to hamper the heart's action, the pulse-rate will be correspondingly accelerated.

Pleurisy is not uncommon ; it is generally dry, and may be unilateral or bilateral. Lobar *pneumonia* occasionally occurs. Rheumatic pneumonia and rheumatic pleurisy are both specially apt to attack the left side of the chest. If the heart is involved, the liability to both these complications is much greater than if the heart escapes. If pericarditis is present, alone or with endocarditis, the liability is much greater than if endocarditis only is present.

Whilst in adults the disease tells most heavily upon the joints, the heart suffers most in CHILDREN. In these, as in adults, the disease may set in abruptly with pyrexia and the usual febrile symptoms. *Arthritis* may be present, but is often slight or altogether absent. *Endocarditis* is common, and, as in adults, tells specially upon the mitral valve. *Dilatation of the heart* takes place, partly, it would appear, through poisoning of the muscle fibres, and partly from actual *myocarditis*. *Pericarditis* may occur at any stage, but is most usual after endocarditis has occurred and the heart is enlarged. It may resemble the acute pericarditis of adults, but is usually of insidious onset and subacute course. The child becomes restless and anæmic, and may vomit ;

the pulse is rapid, and some pain may be complained of in the region of the heart. The inflammation is progressive or recurrent, and may lead to great thickening of the membrane. The child ultimately dies from anæmia, emaciation and exhaustion. Obviously it is often difficult to determine how much of the enlargement of the cardiac dulness is due to dilatation and hypertrophy, and how much to pericarditis.

Rheumatic *nodules* occur in a considerable proportion of cases of rheumatism in children, but seldom in adults. (Such nodules are occasionally seen in osteo-arthritis and other diseases.) They consist of fibrous tissue, and vary in size from a pin-head to a pea. They are attached to periosteum or tendons close under the skin—*e.g.*, over the knuckles, elbows, vertebral spines, and other bony prominences. They may persist for a few days or for many months. They are not, as a rule, tender. When numerous and large, they are likely to be associated with the grave progressive or recurrent forms of pericarditis and endocarditis.

In addition to the arthritis, endocarditis, pericarditis, myocarditis, pleurisy, pneumonia, and subcutaneous nodules, several other manifestations of rheumatism have to be noticed. *Sore throat*, in the form either of tonsillitis, or, more frequently, of catarrh of the fauces and pharynx, is common, and, like pleurisy, occurs in both adults and children. Certain varieties of *erythema multiforme* (especially *e. marginatum* and *e. papulatum*) are met with in rheumatism, occasionally in adults, but much oftener in children. The purpuric form of erythema—viz., *purpura* (*peliosis*) *rheumatica*—also shows a relationship to rheumatism. *Chorea*, which occurs chiefly between five and fifteen years of age, is undoubtedly in many cases a rheumatic manifestation of childhood. *Anæmia*, important in adults, is still more marked in children.

Complications.—Various phenomena formerly described as complications of rheumatism are now recognised as being phenomena of the disease just as much as arthritis, and these have accordingly been included in the above descrip-

tion. Setting these aside, the most dangerous complication is *hyperpyrexia*, though, fortunately, it is a rare one in proportion to the number of cases of rheumatism. *Cerebral rheumatism*, or rheumatism accompanied by delirium and other cerebral symptoms,[1] is, in a large proportion of cases, though not always, accompanied by hyperpyrexia. The typhoid phenomena of rheumatic hyperpyrexia are doubtless due largely to the temperature, at least in cases where the cold bath quickly removes them ; but as hyperpyrexia is not always present in cerebral rheumatism, it would appear that the head symptoms as well as the pyrexia may be due to the action of the rheumatic poison on the nerve centres. Rheumatic hyperpyrexia is far more common in males than in females. It is most frequent between twenty and thirty years of age, and in first attacks of rheumatism. *Orchitis* is an extremely rare complication of rheumatism.[2]

Diagnosis.—The polyarthritis, sweating, and fever, often with some cardiac affection, are generally sufficient for the diagnosis. The principal mistake is to regard osteo-arthritis or gonorrhœal arthritis as ordinary rheumatism, a mistake which is favoured in the former case by the fact that rheumatism is one of the causes of osteo-arthritis. But in *osteo-arthritis* the inflammation does not flit from joint to joint ; the bones and cartilages of the joints undergo alteration ; the second and third metacarpo-phalangeal joints are especially apt to be involved ; the disease is much more obstinate, and if left untreated is apt to be progressive, while cardiac lesions are almost unknown.

In *gonorrhœal arthritis* there is frequently very little constitutional disturbance ; the arthritis is very obstinate, and may give rise to serious limitation of movement in the affected joint ; the inflammation may be long confined to one joint ; fasciæ and tendons with their sheaths often suffer ; and there is usually a history of gonorrhœa.

[1] It should be borne in mind that the salicylates themselves may give rise to delirium.

[2] Bilateral orchitis occurred in a boy of twelve years, and was followed by atrophy of the right testicle (*Trans. Glasg. Path. and Clin. Soc.*, February, 1901).

In *gout*, the patient is generally a male in middle or advanced life ; the inflammation is specially apt to begin in the toe ; the acute attack is characteristic ; and in chronic cases there may be uratic deposits.

Septic arthritis goes on to suppuration ; and rigors, severe constitutional symptoms, and a source of infection may indicate the nature of the disease.

Acute osteomyelitis may be mistaken for rheumatism, since it may begin with pain in the neighbourhood of a joint and with fever, and is sometimes multiple. But osteomyelitis is an affection of the epiphysis rather than of the joint : it does not flit about from joint to joint ; tenderness of the bone may be recognisable, and the constitutional disturbance may be severe.

Prognosis.—Apart from hyperpyrexia, rheumatism is rarely fatal in the *adult*, though it often causes heart disease which in the long-run ends fatally. The ordinary duration of an attack not treated by medicine may be regarded as from one to two weeks, but relapses are very liable to occur, so that the illness is prolonged. Before salicin and the salicylates were introduced, ' six weeks in blankets ' represented the normal duration of treatment, but by modern methods the fever and severe pain are commonly subdued within a very few days.

In the *child*, rheumatism is much more fatal than in the adult, on account of its tendency to cause cardiac dilatation and recurrent or progressive pericarditis. Numerous and large rheumatic nodules are of evil omen.

One attack of rheumatism, so far from protecting, seems to predispose to a subsequent attack. The tendency to involvement of the heart increases with successive attacks.

Treatment.—The patient must rest in bed, preferably between blankets, and should wear a flannel nightdress on account of the sweating. During the febrile period he should be fed on milk. The constipation may be treated by gentle laxatives.

Salicylate of sodium may be regarded as a specific. In an average case, 20 grains may be given to an adult every four or six hours ; in severe cases, the same dose should be

given every two hours. When the fever subsides, as it generally does in a few days, the dose may be reduced to 15 grains every eight hours. Tinnitus aurium shows that the system is fully under the influence of the drug, which should be stopped for a time and then resumed in smaller doses. Hypodermic injections of morphine, or a few doses of Dover's powder, may be desirable at first for the relief of severe pain.

Lees remarks that in the rheumatism of childhood, with little or no arthritis or pain, and with severe cardiac disease, sodium salicylate does as much good as in the rheumatism of adult life. For an adult he recommends 20 grains of sodium salicylate with 40 grains of sodium bicarbonate every two hours during the day, and every four hours at night. For a child of from six to ten years, the corresponding doses are 10 grains of the salicylate and 20 grains of the bicarbonate ; but these should be increased in the course of a few days, if need be, to 15 or even 20 grains of salicylate, with twice the quantity of bicarbonate. A marked deepening of the inspirations (' air-hunger '), when no pericarditis is present, is an occasional symptom of salicylate poisoning, and should lead to the immediate discontinuance of the drug for the time being. The addition of the bicarbonate is supposed to assist in averting this danger.

Instead of the salicylate, salicin may be employed (20 grains every hour or two till the acute symptoms are relieved). Some still trust to alkalies.

The painful joints are to be fomented and wrapped in cotton-wool. Hyperpyrexia requires prompt and sometimes repeated employment of the cold bath ; drugs cannot be relied upon. The treatment of pericarditis will be dealt with in the account of that disease.

After an attack of rheumatism, rest is needed to let the heart regain its tone, and this must be prolonged for weeks, or even months, if the organ has sustained structural damage. Anæmia should be treated by iron. The utmost care should be taken, by the use of suitable clothing and the avoidance of imprudent exposure, to avert a subsequent attack.

18. TUBERCULOSIS.

i. General Considerations.

Definition.—An infective disease, which, local in its commencement, may either remain local or become general; caused by a specific bacillus, and characterised by the development of nodules known as tubercles, which tend to coalesce and to undergo secondary changes, such as caseous necrosis, softening, fibrous transformation, and calcification.

Bacteriology.—The specific bacillus was demonstrated by Koch in 1882. It is a rod whose length is 3·5 μ (half the diameter of a red corpuscle), or somewhat less. It is often slightly curved, and in stained preparations has frequently a beaded appearance. The unstained portions were formerly regarded as spores, but are now looked upon as vacuoles, which may be due to degenerative changes, or may possibly represent an accumulation of reserve material within the bacillus. The bacillus can be grown on various special media at blood-heat, but apart from this is quite unable to multiply outside the animal body, though it may retain its vitality for a long time. It is present in the sputa of consumptives, and in secretions from ulcerating tubercular lesions, as well as in the characteristic lesions in the tissues.

The bacillus is readily detected in the sputum by staining with carbol-fuchsin (Ziehl-Neelsen solution), which consists of fuchsin, 1 part; absolute alcohol, 10 parts; and 5 per cent. aqueous solution of carbolic acid, 100 parts. A suitable portion of sputum is spread on a slide or cover-glass, and, after drying in the air, is fixed by passing three times through the flame of a spirit-lamp. The stain is then poured upon the slide, or the cover-slip is floated on some of the stain which has been placed in a watch-glass. In either case, heat is applied gently till vapour is seen to rise. The preparation is then washed in water, and put for a minute or so in a strong solution of a mineral acid (nitric acid, 33 per cent.; or sulphuric acid, 25 per cent.); this decolourises everything except the tubercle bacilli. The acid is then washed off, and a solution of methylene blue

may be used as a counter-stain. Other bacilli besides that of tubercle possess this 'acid-fast' or acid-resisting property. Among them may be mentioned the bacilli of leprosy, of smegma, of Timothy grass, of butter, and of manure, as well as some streptothrices. These organisms have often been found in milk, butter, and cheese, as well as in normal and pathological secretions of man. Unless an acid-fast bacillus comes from the interior of the body, the staining test is quite inconclusive as to its tubercular nature ; and even if it does come from within the body, the test is not absolutely reliable.

Modes of Infection.—Infection may take place in various ways :

1. Hereditary transmission, brought about by the bacilli entering the fœtal blood-stream through the placenta, is, at least in man, excessively rare.

2. Inoculation is of no great importance in man. The disease usually remains local, and is well known as the anatomical tubercle or post-mortem wart (*verruca necrogenica*) of dissectors. It also occurs in butchers and others who work with dead bodies. Villemin in 1865 first proved the inoculability of tubercle.

3. Inhalation is by far the most common mode of infection in man. A consumptive with active disease in his lungs expectorates enormous quantities of the bacilli, estimated at thousands of millions every day ; and if his sputum is allowed to dry on his handkerchief, bedclothes, or beard, or on the floor or hearth, or on the pavement, the bacilli are wafted about as fine dust, which enters the respiratory passages of healthy and sick alike. In large cities, practically everyone must inhale these organisms, and it simply depends on the numbers inhaled, and the predisposition of the individual, whether he will acquire the disease or not. Thus it happens, though, fortunately, quite exceptionally, that persons living in such intimate contact as husband and wife may acquire the disease from one another through the medium of the dried sputum. The respiratory organs suffer more frequently from primary tuberculosis than any other part of the body.

4. Ingestion is not so important as inhalation in man, though the virus often passes to the lymph glands through the tonsils and the intestine. Milk from a tubercular cow may convey the infection, but this is not likely unless the udder itself is diseased. Moreover, risk from this source can be entirely prevented by boiling the milk. The muscle of animals used as food is rarely tubercular, and even if it should come in contact with tubercular lungs, the danger is averted by thorough cooking.[1] Not uncommonly the consumptive, by swallowing his sputum, infects his own alimentary tract, and the lesions then tend to develop in the agminated and solitary follicles of the intestine.

Wherever a tubercular lesion is situated, the related lymph glands tend to suffer secondarily. It is to be noted, however, that the bacilli are able to pass through mucous membranes without damaging them, and then to give rise to well-marked glandular disease. This is a familiar occurrence in the case of the cervical and mesenteric glands.

[1] At the meeting of the British Congress on Tuberculosis in July, 1901, Koch asserted that the bacillus of human tuberculosis is not identical with that of the bovine disease, and that accordingly there is little or no danger to man from tubercular cattle. Koch, it may be said, based his conclusions first on the results of inoculating bovines with human bacilli, and secondly on his belief that infection through the intestine in mankind is rare. The latter premise is surely altogether at variance with the experience of pathologists. One of the most common lesions found in the post-mortem room is old tuberculosis of the mesenteric glands, and there is no doubt that the infection reached these glands by way of the intestine, exactly as it so often reaches the cervical glands by way of the tonsils. The truth probably lies somewhere between the two extremes. The bovine variety of the bacillus is apparently less virulent towards man than the human variety, though nevertheless distinctly pathogenic. But obviously until Koch's view has been demonstrated to the general satisfaction of scientists, it is the safe course in practice to assume that bovine tuberculosis is a source of danger to mankind.

It is noteworthy that Von Behring is completely at variance with Koch in this matter. He holds that human and bovine tuberculosis are the same disease, and that tuberculosis in man is almost always due to the ingestion of infected milk in infancy. Even the phthisis of adult life he regards as due to ingestion of the bacilli in early childhood, though the infection has remained latent for years or decades.

To many seats of lesion the bacilli must have gained access by the blood-stream, and in many such cases they enter the blood-stream from a pre-existing tubercular focus.

Frequency.—Tuberculosis is the most universally distributed of the fatal diseases of mankind, and it is common among domesticated animals. Yet, while it is a very fatal disease in man, the cases which undergo healing are still more numerous than those which end fatally. Moreover, in countries where attention is given to sanitation, the mortality from the principal tubercular diseases has greatly diminished in recent years. Thus, in England and Wales the annual death-rate per million persons living fell from 1,869 in 1880 to 1,203 in 1903 for phthisis ; from 330 to 194 for tubercular meningitis ; and from 370 to 167 for tubercular peritonitis. On the other hand, it rose from 129 to 178 in the same years for 'other forms of tuberculosis, scrofula.' In 1838, the death-rate from phthisis was over 3,800 ; in 1903, it was 1,203 per million living. During the decade 1881-1890, 1 death out of every 9 was due to tuberculosis, and 1 out of 11 to phthisis. In other words, about 11 per cent. of all deaths were due to tuberculosis, and of each 11 deaths from tuberculosis, 9 were due to phthisis. In the year 1903, 1 death out of every 8·8 was due to tuberculosis, and 1 out of 12·8 to phthisis. Or, to put it otherwise, 11·3 per cent. of all deaths were due to tuberculosis, and 8·3 out of the 11·3 were due to phthisis.

In Glasgow, the phthisis death-rate in the quinquennial period 1860-1864 was 4,094 per million, as against 2,315 in 1890-1894, and 1,712 in 1900-1904.

Predisposing Causes.—High altitudes are relatively free from tuberculosis. Large cities suffer more than country districts. Deficiency of fresh air and of sunlight ; residence over a damp soil ; occupations in which a dust-laden atmosphere is inspired ; any catarrhal condition of the respiratory tract ; measles, whooping-cough, and to a much less extent enteric fever ; diabetes ; influenza ; alcoholism (including alcoholic neuritis) ; congenital heart disease ; aortic aneurysm ; local injury ; and all other conditions, such as deficiency of food, clothing, and rest, which reduce the general vigour of

the body, predispose the individual as a whole, or certain organs in particular, to tubercular infection. Parturition and lactation have an unfavourable influence on women who are already phthisical, and may favour the occurrence of the disease in those previously sound.

Since 1866, the phthisis death-rate in England and Wales has been less among females than among males, and the difference between the two sexes has been increasing. Previous to 1866, the rate was greater among females. A similar inversion of the sexual mortality has taken place in France, and has been attributed to increasing alcoholism among the men. It is probable that another cause—at least, in Britain—is a greater degree of improvement in the domestic than in the occupational conditions of life.

All ages are liable, but different organs are specially susceptible at different ages. Thus, phthisis is specially prevalent from puberty all through the active period of life, whereas tubercular peritonitis causes two and a half times, and tubercular meningitis fully twice, as many deaths in the first five years of life as at all other ages put together. The other forms of tubercular disease, and especially those which involve the bones, joints, and glands, as well as general tuberculosis, are specially common in childhood.

Of late years it has been fashionable to minimise the importance of inherited predisposition ; and cases that seem to exemplify it have been explained on the hypothesis of a very long incubation period, extending possibly over years. Such a theory, however, is not sufficient to account for the facts. No doubt the predisposition in the case of phthisis is sometimes partly attributable to the form of the chest ; but even in the case of scrofula there is an extraordinary tendency on the part of some families to suffer. Similar facts are well known in connection with the exanthemata. The seed is essential, but the soil is scarcely less important.

Morbid Anatomy.—The elementary lesion is the tubercle, a little tumour which is sometimes large enough to be seen by the naked eye, though often of microscopic size. Very often such tubercles are aggregated in clumps, so that their presence is much more noticeable. Such clumps constitute

the ' tubercles ' of gross anatomy. A typical tubercle consists of cells of three kinds : (1) in the central part, one or more branching, multinucleated giant cells ; (2) large epithelioid cells, due, like the giant cells, to proliferation of the fixed cells of the tissues ; and (3) at the periphery, leucocytes (mostly lymphocytes), which have probably migrated from the vessels and represent an inflammatory reaction. Sometimes, however, the giant cells, and sometimes the epithelioid cells also, are awanting in a tubercle. The bacilli are found in the giant cells and epithelioid cells, but, apart from these organisms, there is nothing in the tubercle that is absolutely peculiar to tuberculosis.

A tubercle contains no bloodvessels, and as a rule it undergoes one or other, or a combination, of two further changes, namely, caseous necrosis and fibrous transformation. These changes involve both the tubercle and the surrounding inflammatory products. The necrosis is apparently due, not to the non-vascularity of the tubercle, but to the action of the products of the bacilli. The cheesy-looking or caseous material may afterwards soften and give rise to an abscess if deep-seated, or to an ulcer if superficial ; or it may persist and become infiltrated by lime salts. Fibrous change is less common, but much more curative in its results, than caseation ; it is often seen in the peritoneum.

Tuberculosis almost always gives rise to inflammation. A secondary infection by pyogenic organisms very often takes place in addition.

ii. Acute Miliary Tuberculosis.

General tuberculosis may be either acute or chronic. In the *chronic* form, bacilli from a localised tubercular lesion obtain access in small numbers to the blood-stream, and give rise to secondary lesions in various parts of the body. As the process is chronic, and the lesions are not very numerous, the latter have time to grow to some size before death takes place, and they may give rise to no symptoms, especially if they do not attack the brain.

In the *acute* form, tubercle bacilli gain access to the

blood-stream in enormous numbers, and give rise to countless secondary lesions in many organs. The lungs, liver, spleen and kidneys are almost constantly involved. In some organs, such as the lungs, they can be recognised by the finger as well as by the eye ; whilst in others, such as the liver, they can scarcely be seen by the naked eye, though readily discovered with the microscope. The patient dies while the lesions are recent, so that ' miliary ' tubercles are found everywhere, and the condition is spoken of as acute miliary tuberculosis. The bacilli may reach the blood through softening of a tubercle which has developed in the intima of a vessel, or through the extension of caseation from a tubercular gland to the wall of an adherent vein, or through caseation of a vein in a tubercular gland. The pulmonary veins, thoracic duct, and less frequently other vessels, suffer in this way. The bacilli do not multiply in the blood-stream, but only after coming to rest in some organ. The condition is most common in early childhood. The source of infection in acute general tuberculosis is generally the lung in adults and the bronchial glands in children.

Symptoms.—The clinical features vary much in different cases. There is sometimes a recognisable tubercular lesion of old standing — *e.g.*, in the testicle, lung, pleura, lymph glands, bones or joints, and general symptoms, or symptoms pointing to involvement of one or more distant organs, are in such a case superadded. In other instances, the general symptoms are recognised first, and the disease may perhaps simulate enteric. In such cases, there may or may not be, after a time, evidence of tubercle in the lungs or cerebral membranes. In yet another group of cases, the symptoms of meningitis may first claim attention. If the disease attacks an apparently healthy individual, the starting-point may be deep-seated—*e.g.*, a caseous bronchial gland.

In the *general* or *latent* type, fever is the most important symptom. This is usually considerable, and is remittent or intermittent, and sometimes of inverse type. In the course of a few weeks, the patient dies from coma, or with pulmonary or meningeal symptoms. The diagnosis from enteric may be extremely difficult, but in this form of tuber-

culosis, diarrhœa is exceptional; the temperature is more irregular than in enteric, and is sometimes of inverse type; there are not successive crops of rose-spots; leucocytosis may be present; and Widal's test is negative. Moreover, localising phenomena may develop before death—*e.g.*, in the lungs, brain, or choroid.

The *pulmonary* type is probably the most common. Pulmonary symptoms may or may not have been present for some time before the acute illness sets in. The condition may follow whooping-cough or measles in children. Besides the pyrexia, there are cough, spit, rapid breathing, and cyanosis. In the worst cases, the cyanosis may be extreme; there may be profuse perspiration with an offensive sweetish odour, and the patient may be almost comatose during a considerable part of the illness. The physical signs—apart from any old-standing lesion—are in the main those of bronchitis, with abundant mucous râles over the chest generally; but these râles may possess a coarseness, a resonant quality, or a concentration towards the apices, which should suggest the development of numerous minute cavities. Such physical signs, the history of the case, the discovery of the bacillus in the sputum, or the supervention of localising phenomena elsewhere, will point to the nature of the case.

After the pulmonary type, the *meningeal* type is most common.

Tubercular Leptomeningitis (*acute hydrocephalus*, *basilar meningitis*, *water in the head*, or *water on the brain*) is almost always secondary to tuberculosis elsewhere, and is usually part of an acute general infection. It is much more common in children than in adults. In the former, the source of general infection is often a diseased mesenteric or bronchial gland; in the latter, phthisis is the usual antecedent, though this may have escaped detection. Tuberculosis of the pleura, bones, joints, brain, or genito-urinary organs may also be the source of infection. When meningeal symptoms set in, they generally dominate the case, and other evidences of a general invasion may be inconspicuous or quite overlooked.

Morbid Anatomy.—The changes are best marked, as a

rule, at the base of the brain. The exudation is specially well seen in the space bounded by the crura cerebri and the optic tracts, and it extends thence into the Sylvian fissures and perhaps along the pons and bulb. The spinal membranes are frequently involved. The exudation is opaque, but not purulent. Tubercles may be seen in the pia mater in the form of little white nodules, which consist of aggregations of round cells, and are often connected with the adventitia of the arteries. The media and intima may be invaded, so that the lumen is narrowed. The ventricles are distended with fluid, and the increased pressure may cause flattening of the convolutions on the convexity. The brain substance in contact with the inflamed membranes is often infiltrated with round cells, and may contain tubercles (*meningo-encephalitis*).

Symptoms.—The acute symptoms are often preceded for some weeks by prodromes or premonitory phenomena, of which the principal are loss of flesh and strength, restlessness, and headache. These are attributable to the general invasion of the system, though the headache may be due to development of tubercles in the membranes before actual inflammation has been excited.

Commencing with the severe symptoms, three stages are sometimes described. The onset is characterised by intense headache, vomiting, pyrexia, and often convulsions. The headache may induce the 'hydrocephalic cry.'[1] The bowels are constipated. The head is often retracted. This is the stage of *irritation* or excitement.

The stage of *depression* or compression which follows may give rise to false hopes, for the temperature may be normal, vomiting absent, and sleep almost constant, though the patient can be roused to take nourishment. But the pulse seldom shares in the improvement, and symptoms pointing to palsy of various cranial nerves occur. There is often slight optic neuritis, the abdomen is retracted (boat-shaped, 'carinated'), and the respirations are disturbed. The pulse is slow and irregular, and often out of correspondence with the temperature, which itself varies much in different cases.

[1] Or 'cephalic cry'—a sudden loud scream. It may occur also in intracranial tumour.

The *tache cérébrale* may be well marked (an exaggeration of the congestion of the skin which follows the stroke of a finger-nail); it is not, however, peculiar to this disease.

In the stage of *paralysis* and coma, the child cannot be roused, the limbs may be rigid or weakened on one side, the pupils are dilated, the pulse is rapid, and the evacuations are passed into the bed. There may be, at the close, either hyperpyrexia or a subnormal temperature.

The usual duration of the acute illness is two or three weeks.

In the adult, the disease is very similar to that in the child. General convulsions, however, are uncommon; delirium usually sets in earlier; in young women hysterical symptoms may occur at the outset; and recovery is less rare. If the disease affects specially the convexity, the diagnosis may be difficult for a time. The cranial nerves may escape, and cortical symptoms, such as aphasia, may occur early and perhaps suddenly.

Diagnosis.—This depends on the acute or subacute onset of head symptoms with fever, often after premonitory symptoms, and in a subject known to be tubercular. The most important symptoms are severe headache, which persists after delirium sets in; causeless and repeated vomiting; optic neuritis of slight degree, and palsies of one or more cranial nerves. Kernig's sign may be present, and lumbar puncture may be tried. In the case of a quickly growing *intracranial tumour*, which might give rise to some difficulty, any palsy of the limbs develops more gradually, and optic neuritis is often intense; uncertainty may, however, continue for a week or two. In *fevers* like enteric, headache ceases when delirium begins. In *hydrocephaloid* (a more or less comatose state which sometimes supervenes in young children exhausted by diarrhœa), the fontanelle is depressed, fever and localised paralysis are absent, and there may be a history of diarrhœa or some other cause of cerebral anæmia.

In a few cases of acute miliary tuberculosis—*abdominal* or *typhoid* type—the symptoms are highly suggestive of enteric fever. In addition to the more general symptoms of typhoid, there may be abdominal distension and tenderness, diarrhœa,

splenic enlargement, a few rose-spots, and even, it may be, a positive Widal's reaction. This type may last for six or eight weeks. The diagnosis from enteric may be for long impossible. The presence of pre-existing tubercle or of tubercles in the choroid and an inverse temperature are in favour of tuberculosis.

Prognosis.—In acute miliary tuberculosis, no hope of recovery can be entertained. Recovery from tubercular meningitis is generally, though not universally, regarded as extremely rare. When it does occur, the presumption is that the bacilli are not disseminated all through the system.

Treatment.—This is almost purely symptomatic—nursing, feeding, regulation of the bowels, and cold sponging if the fever is high. In tubercular meningitis, the head should be shaved, and ice should then be applied. Mercurial ointment, with or without an equal proportion of iodine ointment, should be rubbed into the scalp and other parts, at intervals of a few hours, until the mouth just begins to be affected. Lumbar puncture has had a curative effect in at least one instance. For headache, phenazone or phenacetin may be administered; and for vomiting, sinapisms over the stomach and back of the neck, with ice and chloroform water internally, are appropriate measures. Opium should be avoided, because of the tendency to coma.

iii. Tuberculosis of the Cardiovascular System.

A few miliary tubercles may be found in the *myocardium* in acute miliary tuberculosis, but otherwise this tissue is very rarely involved.

In rare cases of phthisis there is *endocarditis*, which results usually from a mixed infection, but is occasionally due to the tubercle bacillus.

Tubercular *pericarditis* is referred to under Tuberculosis of Serous Membranes (p. 132).

In organs such as the lungs, which are the seat of tubercular disease, the walls of the *arteries* and *veins* may become involved, either by an acute infiltration, or by a more chronic process which goes on to caseation. Thrombosis may occur,

or softening may take place; and in the latter case, the bacilli may enter the blood in large numbers. In the acute miliary disease, tubercles may develop in the intima or in the adventitia of bloodvessels.

iv. Tuberculosis of the Respiratory System.

Tuberculosis of the Larynx (*Tubercular Laryngitis, Laryngeal Phthisis, Consumption of the Throat or of the Windpipe*), though in rare cases primary, is in the vast majority of cases secondary to pulmonary disease. It may occur when few or no changes can be detected in the lungs; the diagnosis may then be difficult, while it is at the same time of special importance. As a rule, however, tuberculosis of the larynx is a late phenomenon in pulmonary phthisis. It is present in about 40 per cent. of cases of late phthisis, and is attributable to infection of the larynx by the bacilli in the sputum.

Morbid Anatomy.—The tubercles first develop beneath the epithelium of the mucosa. Caseation ensues and leads to ulceration, which spreads. The principal seats are the vocal cords and the interarytenoid fold, but the ary-epiglottic folds, the epiglottis, and occasionally parts further off, may suffer.

Symptoms.—There is hoarseness or complete loss of voice. In the early stages, there may be some discomfort at the seat of disease, but when ulceration sets in, and especially when the epiglottis and ary-epiglottic folds are involved, there is apt to be severe pain on swallowing, as well as mechanical dysphagia. In advanced cases, food may get into the trachea. The cough is altered in character when the cords are much affected.

The laryngoscope shows in the early stage pallor of the mucous membrane. Later on, there is thickening from infiltration, very often first in the interarytenoid space. Then the diseased parts ulcerate, and acquire a worm-eaten aspect.

The mobility of the vocal cords is often impaired mechanically by the infiltration. But immobility may be due to paralysis of one or both of the recurrent nerves. In laryngeal

phthisis, the right nerve is much more frequently paralysed than the left, and it has been supposed that this is due to the right recurrent nerve being involved in pleuritic thickening at the right apex. When the left nerve is paralysed, it is perhaps owing to pressure by enlarged bronchial glands.[1]

Diagnosis.—Chronic laryngitis persisting for months should be regarded with suspicion. The sputum, and in case of ulceration some of the secretion, should be examined for bacilli. In *syphilis* there is little or no pain, the voice is hoarse rather than lost, ulceration is commonly less chronic than in tubercle, and the disease may be expected to yield to treatment. *Malignant disease* rarely occurs before forty years of age, and is characterised by constant pain ; whereas in tuberculosis there is but little pain so long as the patient is not eating, coughing, or otherwise disturbing the parts.

Prognosis.—This is generally unfavourable, partly because advanced lung disease is usually present. If, however, there is little or no lung disease, and the laryngeal disease is early and localised, there is some hope.

Treatment.—General measures should be employed, as in the case of phthisis. The ulcers should be cleansed by some weak astringent or antiseptic spray introduced by an atomiser.

To destroy the tubercular tissue in early cases, the ulcer is first painted with a 10 per cent. solution of cocaine, and then carefully scraped by a curette. Thereupon lactic acid is thoroughly rubbed in by the cotton-wool brush, in a strength which is gradually increased from a 20 per cent. solution up to the pure acid. This is repeated once every day or two.

In cases which cannot be treated by such radical measures, a 4 per cent. solution of cocaine may be applied by the atomiser to relieve the pain and permit swallowing ; or a powder of iodoform with a little morphine may be insufflated several times a day after the ulcers have been cleansed by the spray. Another useful spray is 20 per cent. menthol in parolein ; or the same strength of menthol in olive oil may

[1] In aortic aneurysm, which is sometimes associated with laryngeal palsy, it is generally the left recurrent nerve which suffers.

be rubbed in by the cotton-wool brush. Codeine lozenges may be sucked by the patient to diminish the local irritation.

Tobacco, alcohol, spices, etc., should be avoided, or used sparingly. If swallowing is difficult or painful, the food should take the form of thick, bland liquids, and should be gulped rather than sipped. When the epiglottis is destroyed, so that liquids tend to enter the larynx, the patient should suck them through a rubber tube while lying on his face.

The *trachea*, and less frequently the *bronchi*, may be the seat of tubercular ulceration in advanced phthisis, but no distinctive symptoms are recognised.

Tuberculosis of the Lungs (*Phthisis Pulmonalis*,[1] *Consumption*[2]).—In England and Wales, in recent years, about eight deaths out of every hundred have been due to phthisis, and other three have been due to other forms of tubercular disease ; in other words, nearly three-fourths of the deaths from tuberculosis are due to phthisis (see p. 104). Apart from acute miliary tuberculosis, which has been already described, tuberculosis of the lungs may be either acute or chronic. The acute type may be either pneumonic or broncho-pneumonic (p. 115) ; and the chronic type may be either catarrhal, caseous and ulcerative (p. 116), or fibroid (p. 126).

(*a*) *Acute Pneumonic Phthisis* (*Confluent form of Acute Pulmonary Tuberculosis*, *Scrofulous Pneumonia*, *Tubercular Caseous Pneumonia*).—This disease is very apt to be mistaken for lobar pneumonia of the apex. It may involve one lobe or almost the whole of one lung. The anatomical condition (lobar consolidation, scanty pleural exudation,

[1] *I.e.*, wasting, or consumption (of the body generally), associated with pulmonary disease.

[2] Laennec, who taught the unity of different types of tubercular lesion ; who, by his discovery of auscultation, enabled us to recognise the presence of tubercle in the lungs during life ; and who gave the greater part of his professional life to the study of phthisis, himself died of phthisis in 1826. He attributed his illness to an autopsy wound, though he did not die till many years had elapsed after the inoculation.

etc.), the physical signs (dulness over one lobe, increased vocal fremitus, enfeebled and bronchial respiratory murmur, and bronchophony), and the symptoms (sudden onset of pain in one side, with short cough, scanty and rusty sputum, fever, and rapid respiration), constitute so exact a picture of lobar penumonia that for a week or so the diagnosis during life may be impossible.

Anatomically, however, the appearance of hepatisation is seen, on careful inspection, to be due to the aggregation of tubercles, although in many cases croupous inflammation is also present, and contributes to the consolidation. Examination may disclose an older focus of caseation or softening, from which the acute infection of the lobe took place, probably by aspiration.

Clinically, a careful observer may note grounds for suspicion. The patient may have been in poor health previously ; the sputum, which may be rusty or bloody, may contain tubercle bacilli and elastic tissue ; and the daily fluctuation of the temperature may be greater, and the tongue less dry, than in ordinary croupous pneumonia. After a week or ten days, there is still no sign of a crisis. Hectic fever, profuse sweating, emaciation, and abundant purulent expectoration are observed ; and the physical signs, which include coarse, clicking râles, point to the development of cavities in the lungs.

Death may take place as early as the second week, before softening has occurred. But, as Douglas Powell has shown, cases of this type tend to undergo arrest by elimination of the caseous material, by cicatricial contraction of the cavities formed, and by compensatory hypertrophy of the opposite lung. If the patient can survive the suppurative processes, with the attendant emaciation, hectic fever, and sweating, and if the other lung is not infected by insufflation of the bacillus-containing material, the symptoms, after a period of from six weeks to four months, gradually subside, the affected side shrinks, the opposite lung enlarges, the cavities contract, and in favourable cases a good recovery is made.

(*b*) *Acute Broncho-pneumonic Phthisis* (*Acute Tubercular*

Broncho-pneumonia, Disseminated form of Acute Pulmonary Tuberculosis, Florid Phthisis, Galloping Consumption).—This disease is more common than the pneumonic form, especially in children. It begins in the smaller bronchial tubes, but the catarrhal process extends into the alveoli. The catarrhal products tend to become caseous, so that the tubes are blocked with caseous matter. The affected areas have a reddish-gray appearance before they become caseous. Still later, the caseous matter softens, so that little abscesses develop in the consolidations, and have their walls lined by caseous matter. As the disease starts from the bronchi, the consolidations have a lobular arrangement. Small cavities may coalesce to form larger ones, and in other parts of the lungs there may be miliary tubercles. The overlying pleura is generally covered by exudation, but sometimes it is undermined by a softening near the surface of the lung, and perforation occurs. Emphysema and collapse may be associated with this disease.

The onset is often gradual, but may be rapid. In children, the disease frequently follows measles or whooping-cough. Shivering, malaise, loss of flesh and strength, cough, anorexia, and sometimes hæmoptysis, are the early symptoms. The physical signs are those of broncho-pneumonia—dry and moist râles, indicating bronchitis, with scattered patches in which bronchial or tubular breathing, bronchophony, dulness on percussion, and increased vocal fremitus, indicate little areas of consolidation. There is considerable fever. Tubercle bacilli and elastic tissue may be found in the sputum. Death may occur within a few weeks, or the case may undergo considerable improvement and pass into a chronic phase.

(*c*) *Chronic Ulcerative Phthisis* (*Chronic Catarrhal* or *Caseous Phthisis*).—This is by far the most common form of pulmonary tuberculosis. Some of the cases, more rapid in their course than others, deserve to be called subacute.

Etiology.—This has been discussed at some length under the heading of General Considerations (p. 104). But while the importance of inhalation as a mode of infection in phthisis has been already pointed out (p. 102), it remains

to be said that in young children pulmonary tuberculosis is often, and perhaps in the majority of cases, due to extension from tubercular bronchial glands. The pulmonary group of bronchial glands are practically embedded in the lung tissue, and tubercular disease in them is apt to spread into the surrounding lung substance. Even in the adult tuberculosis of the supraclavicular and axillary glands may be the immediate precursor of phthisis. A softened bronchial gland may also burst into the trachea, a main bronchus, or a smaller bronchial tube, and so infect the lungs.

Morbid Anatomy.—An immense variety of conditions is met with in the lungs in a case of this disease. Fibrous transformation is always present in addition to caseation, and as new foci of disease are constantly arising through secondary infection from older ones, all stages can usually be observed in the same lung. The disease begins in the fine bronchi, whose walls may become ulcerated, and it spreads thence to the alveoli.

The disease almost always begins, and is found most advanced, in the apex of the upper lobe. Its favourite seat of commencement is about an inch below the top of the lung. The apex of the lower lobe on the same side, and then the apex of the opposite upper lobe, tend to become affected soon afterwards. Both lungs are almost always diseased at the time of death, but the changes are more advanced in one than in the other. The special liability of the apex is probably due to relatively inefficient ventilation of that part, whereby the bacilli have longer time to settle in the tissues. This inefficiency is due partly to limited expansion of the upper part of the chest, and partly to the very indirect course which the respired air is compelled to take in passing from the trachea to the apex, and *vice versâ*. In little children, where the pulmonary changes are the result of extension from softened bronchial glands, there may be advanced disease in the lung around its root, while the apex and more peripheral parts are healthy, or at most the seat of scattered tubercles. Even in adults, however, pulmonary tuberculosis is not necessarily most advanced at the apex.

Cavities (*vomicæ*) are almost always present. They are of all sizes, and one of them may involve almost a whole lobe. Old healed cavities are lined by fibrous membrane with a smooth surface; but those that are usually seen have irregular walls, roughened by softening caseous material, or lined by a more distinct pyogenic membrane. They contain pus, caseous matter, broken-down lung tissue, etc. They may be very irregular in form, and are often crossed by fibrous tissue septa or strands containing obliterated vessels.

Masses of caseous material may be recognised, probably surrounded by miliary tubercles, which in their turn will become caseous. If the cheesy matter does not proceed to softening, fibrosis may go on around it to such an extent as to enclose it in a capsule, after which it tends gradually to become infiltrated with lime salts. This is a common mode of healing in tuberculosis.

The features of tubercular broncho-pneumonia may also be seen, the small bronchi and their alveoli being full of catarrhal products which, like themselves, undergo caseation.

The disease, in a more acute phase, may extend from the mucosa of the fine bronchi through their walls into the surrounding tissue, and along with this there may be acute (croupous) inflammation of the neighbouring alveoli.

Miliary tubercles may be found in various parts of the lungs. The small bronchi and alveoli of particular areas become infected by aspiration, and the lymphatic channels become infected by the current within them. Bronchiectasis is sometimes observed, especially when there is much fibrosis; and emphysema is also common. Aneurysms of branches of the pulmonary artery are sometimes found in cavities.

The pleura is adherent over the diseased portions of lung. Pleural effusion may be present on one or both sides. Pneumothorax may be present.

The bronchial glands, larynx, and intestines are often tubercular. There may be evidence of a chronic or of an acute general infection by the tubercle bacillus.

The heart is often atrophied; and amyloid degeneration of

viscera, fatty degeneration of the liver, *fistula in ano*, and marasmic thrombosis are sometimes noted.

Symptoms. — These include cough and expectoration, hæmoptysis, loss of flesh and of strength, night-sweats, anorexia, dyspepsia, anæmia, and in women amenorrhœa ; but one or several of these phenomena may be absent. The *onset* is usually insidious, and varies greatly in different cases. Often the patient appears to be suffering from an ordinary cold, which, however, does not pass away after the usual time. In other cases, spitting of blood, or the pain of a dry pleurisy, or indigestion, may first attract attention.

Cough is one of the most constant symptoms all through, and in the later stages is apt to cause vomiting. The sputum is at first mucous or muco-purulent, as in bronchitis, but it tends to become more and more purulent. When suppurating cavities are present, the sputum may consist almost entirely of pus, and when this is expectorated upon a flat surface, it assumes a coin-like shape, and is hence described as nummular. Such sputa often sink in water.

Blood may be present in the expectoration at any stage of the disease. Blood which comes from the lungs is usually bright red and frothy. It stains the spit for some hours or days after the onset of the hæmoptysis.

Hæmoptysis is often the first symptom, or one of the earliest. In such cases, the physical signs may be normal, and bacilli may or may not be present in the sputum. Sometimes the hæmorrhage follows exertion, but this is not the rule, and the onset is frequently by night.

It has long been thought that there is a *phthisis ab hæmoptoë*, *i.e.*, that the presence of blood in the air-passages, from whatever cause, can give rise to inflammatory processes which pass on to caseation ; in other words, that hæmoptysis is occasionally a cause of phthisis. It is very doubtful if such a thing ever happens. The expression 'hæmorrhagic' is now applied to a variety of phthisis in which profuse and repeated hæmorrhages are the principal feature. This type is five times as common among men as among women. It occurs later in life than the ordinary

form, and is seldom due to inherited tendency. The hæmorrhages may recur at intervals of days, months, or years. Abnormal physical signs may be few or altogether absent; but if the bleedings occur very frequently, the case may come to present the symptoms and signs of ordinary phthisis. It is supposed that the hæmorrhages are due to erosion of considerable vessels by tubercle bacilli.

Hæmoptysis is described as early or late, according as the blood comes from a commencing or from an advanced lesion in the lung. As new lesions continue to form throughout the disease, *early* hæmorrhage may occur at any time in its course. This is due to diapedesis, from local hyperæmia, or possibly in part to softening of the capillary walls in the earliest stage of the disease. *Late* hæmorrhage results from rupture of an aneurysm, and is often severe and sometimes fatal. A branch of the pulmonary artery passing along the wall of a cavity, or across it, is usually obliterated by thrombosis, but sometimes this is not so, and the inflammatory or tubercular process softens the vessel wall. The latter, being deprived of its normal support, bulges into the cavity and is apt eventually to rupture.

Tubercle bacilli are often to be found in the sputum, sometimes in enormous numbers. They are never found in the expectoration, except in tubercular cases. *Elastic fibres* from the walls of the alveoli may also be present, but these are sometimes found in bronchiectasis. If elastic tissue is not easily found by examining some of the more opaque parts of the sputum with the microscope, some quantity of the expectoration should be boiled with an equal quantity of caustic soda solution (20 grains to 1 ounce) until the mixture is quite liquid, but not any longer. The liquid should then either be allowed to settle for some hours in a conical glass, or be centrifugalised, after which the sediment may be removed by a pipette for examination under the microscope. The appearances are scarcely characteristic unless the alveolar arrangement of the fibres can be recognised.

Dyspnœa is seldom marked except on exertion. Pain in the chest is not uncommon, and may be the first thing com-

plained of. It is attributable to pleurisy. Gastric symptoms are common.

Loss of flesh and strength is the rule, and is often very marked ; but under treatment by overfeeding, much weight may be gained, even though the patient is on the whole losing ground.

Fever is almost always present in progressive cases, and is usually intermittent or remittent in type, and occasionally inverse. When the daily paroxysm of hectic fever is setting in, the cheeks are often flushed, and, as it passes off in the early morning, profuse sweating sets in, and annoys and weakens the patient. When cavities are present, there is no doubt that the fever is partly due to septic absorption. In very chronic cases, and in cases that are recovering, the temperature may not rise above normal, but may at one part of the day fall considerably below the normal. The night-sweats may give rise to sweat rashes (sudamina, etc.). In progressive cases, a tap on the skin over the costal cartilages will give rise to local elevation of the skin (*myoidema*), owing to contraction of the intrinsic cutaneous muscle ; and the fibres of the pectoral muscle will also contract when struck. The ends of the digits often become clubbed and the nails curved (*Hippocratic fingers*). Anæmia is present, the blood-plates are increased, and in women amenorrhœa is very common.

Physical Signs.—In earlier days, three stages of phthisis were recognised : consolidation, softening, and excavation. It is allowable, though unnecessary, to recognise these from the anatomical point of view ; but from the clinical aspect, the terms are quite misleading ; first, because all stages are often present in a lung simultaneously, and, secondly, because a patient may be much better, and have a better prospect of recovery, with a cavity in his lung (third stage) than if he has a mass of caseous material in place of that cavity (second stage). Excavation is one of the modes of healing.

Inspection may show that the patient has a flat or a winged chest (congenital deformities), though any kind of chest may suffer. There may be flattening and deficiency of movement on the affected side.

Palpation will also reveal any deficiency of movement. Vocal fremitus[1] will be increased over an area of consolidation, and diminished over a pleural effusion.

Relative dulness at the apex is a common early sign, and is attributable to consolidation of lung, to thickening of pleura, or to both of these causes. Sometimes, however, just as in pneumonia, consolidation gives rise to a tympanitic note. A cavity of some size, if superficial, will give a tympanitic note, unless it be filled with secretion, in which case it will yield dull percussion. A cracked-pot sound (*bruit de pot fêlé*) is obtained on percussing over a superficial cavity which communicates freely with a bronchus. It is obtained best if the patient keeps his mouth open.[2]

Enfeeblement of the respiratory murmur, bronchial breathing (characterised by hollowness of the respiratory murmur with prolongation of the expiratory portion), bronchophony, fine moist râles and scanty pleural friction are frequent early signs. Indeed, one or two of these phenomena detected over the apex may be the earliest physical signs in the chest. As the disease progresses, moist râles become audible, or, if heard earlier, become coarser and distinctly clicking in character. In a growing cavity, they become more and more metallic. Over a superficial consolidation, the breath sound is tubular, and pectoriloquy is present. Over a cavity of some size, the breath sound is cavernous. A very large cavity may yield amphoric phenomena, such as amphoric echo, metallic tinkle, the bell sound, or succussion. Pleuritic friction may be heard over the diseased portion of lung or elsewhere in the chest.

Complications.—These include tuberculosis of the larynx, intestines, and meninges ; pneumonia (which may cause death, or be recovered from) ; emphysema ; gangrene of the lung ; pleurisy with, but more commonly without, effusion ; pneumothorax ; venous thrombosis, especially in the left thigh ; catarrh and often dilatation of the stomach ; dys-

[1] In the healthy chest, vocal fremitus is often slightly greater, and resonance less over the right than over the left apex.

[2] The cracked-pot sound is yielded by healthy chests in childhood.

pepsia ; diarrhœa ; *fistula in ano ;* nephritis ; mental symptoms (euphoria or a sense of well-being—the *spes phthisica* —being very common, though, on the other hand, suicidal melancholia may be met with) ; tinea versicolor ; and amyloid degeneration of viscera. The tubercular infection may become general.

Diagnosis. — In advanced cases, with symptoms and physical signs well marked, there is no room for doubt. But the patient may complain of almost no symptoms, although the physical examination points to advanced disease ; and, conversely, symptoms may be well marked, though there are no distinctive physical signs. The presence of tubercle bacilli in the sputum is certain evidence, and the presence of elastic fibres is strongly suggestive. Hæmoptysis should always be regarded with suspicion, until a source can be found for it outside the lungs. Similarly, a bronchial catarrh in which the physical signs are concentrated at the apex must be carefully watched.

Prognosis.—This must be founded largely on the observed course of the disease. Rapid progress of the disease as evidenced by severe symptoms is very unfavourable ; while an absence of symptoms, even though physical signs are well marked, gives good ground for hope. Parturition ; alcoholism ; insufficiency of food, clothing, sunshine, and air ; tuberculosis in other organs ; and profuseness of expectoration, with abundance of bacilli and elastic fibres in it, influence the prognosis unfavourably. On the other hand, absence of pyrexia, a good digestion, limitation of the disease to the apex of one lung, and good social circumstances, are favourable. Among the upper classes, the patient usually survives for a considerable number of years. Among the humbler classes, the expectation of life is considerably shorter.

Evidences of healed pulmonary tuberculosis are met with in about 20 per cent. of the post-mortem examinations which are made on the bodies of persons who have died of non-tubercular diseases ; chiefly in the form of fibrous, caseous, or cretaceous nodules in the apices of the lungs, but occasionally in the shape of a cavity lined by a beautifully smooth

membrane. It is probable that in a large proportion of such cases the lesion was not recognised during life.

Treatment.—The food should be light, nutritious, varied, and in as great quantity as the patient can digest. If systematic overfeeding is aimed at, raw eggs between meals and finely-pounded raw meat may be added in considerable quantity to the more ordinary diet. If the stomach is irritable, it may be necessary to give small meals every two or three hours. The bowels must be regulated, and if there is diarrhœa, certain kinds of food must be withheld until this is checked.

The clothing should be such as to protect from sudden changes of temperature without oppressing the patient. Flannel, therefore, should be worn next the skin.

Cases with considerable fever, sweating, and other marked symptoms, are best kept in bed ; but where the disease is very slowly progressive, and fever is almost or quite absent, the patient should take regular exercise of a kind and amount which will not cause the least fatigue.

Next to food and clothing, and almost equal to them in importance, are fresh air and sunlight. The more fresh air the patient can get, the better for him. The modern sanatorium treatment aims chiefly at keeping the patient constantly in the open air, and, in some cases, at systematically overfeeding him in addition. If he is covered with a sufficiency of clothing, he may remain out of doors all night in dry, calm weather, and be none the worse, but all the better for it. At some sanatoria, there are shelters which can be turned so that the closed side protects the inmates from the wind. Febrile cases may lie in their bedrooms with windows and doors wide open. Wealthy patients who are strong enough may spend the winter abroad in a climate less moist and changeable than our own. A sea voyage may be very advantageous in an early stage, or in completing the cure of an arrested attack ; but obviously patients who are not fit to go on deck should not attempt a voyage ; and persons without means or friends, or a certain assurance of suitable employment, ought to stay at home rather than go to Australia or South Africa.

Of medicines, the most important are cod-liver oil and creosote. The former is to be regarded very much as a food, and is given in doses of 1 to 4 drachms thrice daily. It should be taken as the last course at meals, and a dose may also be taken at bedtime. Creosote is best given in emulsion, and the following combination rarely disagrees: ℞. Creosoti purif., ♏i.; mucil. acac., ʒi.; sp. chlorof., ♏ii.; aq. menth. pip., ad ʒvi. M. Sig.: ʒss. t.i.d. p.c. This dose represents 5 minims of creosote to begin with, and the dose should be increased before long to 10, 15, or even 40 minims for an adult. Capsules of creosote should be avoided. If for any reason creosote does not suit, guaiacol carbonate should be given. This is a tasteless and odourless salt, which may be taken even by children for indefinite periods; 5 to 40 grains may be given thrice daily to an adult. Treatment by compressed air appears to be helpful in some cases; and the air which is used may be saturated with creosote.

Arsenic should be given in chronic cases with marked *anæmia*. Stomachic tonics and bismuth are often indicated where the appetite is poor and the stomach is out of order. The longer *cough* mixtures can be avoided the better, since those that relieve the cough are very likely to impair an already weak digestion. Dilute hydrocyanic acid, however, tends to diminish both coughing and vomiting; it may be combined with an alkali in a bitter infusion. Chlorodyne, liquorice lozenges, and syrupy preparations, such as the syrup of Virginian prunes, given about bedtime, are among the least harmful sedatives. Relief may sometimes be obtained by the inhalation for half an hour at a time of creosote, spirit of chloroform, compound tincture of benzoin, carbolic acid, etc. A few drops of two or more of these are put on the sponge of a metal inhaler. *Sweating* at night should be treated by $\frac{1}{80}$ of a grain of sulphate of atropine given at bedtime. Occasionally, if the sweating is severe, two such pills may be given, but if this dose is continued, it is apt to cause unpleasant dryness of the mouth. For *hæmoptysis* the treatment includes absolute rest in bed, morphine hypodermically, feeding with cold substances, restriction of liquids, applications of ice to the

affected part of the chest, ice sucked in the mouth, and turpentine (10 minims every hour for five doses, in capsule, milk, or syrup). Ergot is to be avoided. Salts of calcium increase the coagulability of the blood, and are of great value in preventing hæmorrhage from the lungs or elsewhere. The chloride of calcium (5 to 15 grains), or the saccharated solution of lime ($\frac{1}{2}$ to 1 drachm) may be given thrice daily in milk. *Diarrhœa* may be treated with bismuth, with lead and opium pill, or with enemata of starch and laudanum.

External applications are often called for in the form of liniments for the relief of pain, or in the form of blisters or liniments for the purpose of counter-irritation. Poultices and hypodermic injections of morphine (preferably local) will be needed if the pain is severe; or the side may be strapped.

The curative value of tuberculin, of antitubercular sera, and of intravenous injections of antiseptics, is still so far from being established that these agents need not be further mentioned here.

The patient must be instructed never to swallow his sputum, and care should be taken that the latter is never allowed to become a source of danger to the patient or to others by drying, or in any other way. If the patient is going about, he should have a pocket spittoon, and the contents should be emptied at least once a day, either into the fire or down the water-closet. The spittoon should then be cleaned with boiling water. If he is in the house, he can spit into paper or rags, which should be burned immediately. In hospitals, a disinfectant solution is put into the spittoon before it is placed beside the patient.

(*d*) *Fibroid Phthisis.*—This is the relatively uncommon form of pulmonary tuberculosis in which fibrous transformation predominates over caseous necrosis. There is a good deal of uncertainty as to its etiology. In some cases—perhaps owing to individual peculiarity—the tubercle bacilli cause fibrosis from the outset. In other cases, when ordinary caseous phthisis becomes very chronic, it assumes the aspect of fibrosis. And, again, chronic irritation, as in pneumonokoniosis, may cause fibrosis, and the bacilli may either enter

from the first along with the dust or settle in the lung after it is damaged. It is possible, also, that a chronic tubercular pleurisy may lead to involvement of the interstitial tissue of the lung.

Morbid Anatomy.—The affected lung is shrunken and indurated. It contains a great excess of fibrous tissue in the form of white bands, which are pigmented and contain tubercles. There is marked dilatation of bronchi. The pleura is firmly adherent, and often greatly thickened. Compensatory emphysema is present. The disease may commence at the apex of the lung or at the base, and may long remain unilateral.

Symptoms.—The most important are cough, which may be paroxysmal ; occasionally hæmoptysis ; loss of flesh and strength ; shortness of breath on exertion, and pain in the side. There is little or no fever. When bronchiectasis develops, the sputum may become fœtid. The disease is very chronic, and is long confined to one lung ; but ultimately the opposite lung is apt to be infected with miliary tubercles. Moreover, amyloid disease of the viscera frequently supervenes. Under favourable conditions, however, such patients may survive for many years with their one lung ' gone.'

The principal physical signs are shrinking and loss of movement on the affected side, drawing over of the heart towards that side, dulness on percussion, and other signs of consolidation and cavity. Moist râles are absent or scanty, but pleural friction may be audible.

Treatment.—The patient should be warmly clad, must avoid exposure to wet, and ought to have regulated exercise in the fresh air. He should take cod-liver oil in winter. Small doses of chlorodyne might be taken at bedtime to allay severe cough, but it is better if such things can be avoided altogether.

Tubercular *pleurisy* is referred to on p. 132.

v. Tuberculosis of the Alimentary System.

The TONGUE is occasionally affected in persons suffering from phthisis or some other form of tuberculosis. Tubercles

develop in the mucosa on the dorsum or edge of the tongue, become caseous and ulcerate, and the disease tends to spread slowly.

The condition may be difficult to distinguish from syphilis or epithelioma, but it may be noted that it does not yield to potassium iodide, and that sometimes definite tubercles can be seen or felt. A scraping may be removed for microscopic or bacteriological examination, or for an inoculation test. In favourable cases, excision may be practised; otherwise cleansing and soothing applications should be used.

The TONSILS are occasionally the seat of tuberculosis, which goes on to caseation and ulceration. It is probable that the common tuberculosis of the cervical glands is due in most cases to infection by bacilli which pass through the tonsils without actually attacking the latter, and in spite of the resistance offered by these great accumulations of lymphoid cells.

The *palate* and *pharynx* may be infected in connection with pulmonary and laryngeal tuberculosis. When the pharynx is ulcerated, deglutition may be very painful.

Tuberculosis of the *stomach*, apart from general miliary infection, is rare, probably because the gastric juice inhibits the growth of the bacilli.

The INTESTINE is often the seat of tuberculosis in consumptives through swallowing of the expectoration. The disease frequently occurs primarily in children, but very rarely in adults.

Morbid Anatomy.—The region of the cæcum and ileum suffers most, the liability diminishing both upwards and downwards from these parts. Peyer's patches and the solitary follicles are the special seats of the tubercles. Caseous necrosis and ulceration take place. The ulcers tend to spread around the bowel rather than along its axis; the edges are infiltrated; and tubercles may be seen on the serous aspect of the bowel (three points of distinction from the typhoid ulcer). Stricture of the bowel may occur. Perforation is not so common as in enteric. The mesenteric glands are constantly involved.

Symptoms.—These are not characteristic. The most

suggestive is diarrhœa in a person known to be tubercular ; but such diarrhœa may be due to amyloid disease or to various other conditions. On the other hand, constipation may coexist with tuberculosis of the intestine. Since the appendix as well as the cæcum is often the seat of tubercular ulceration, changes may take place in this region which raise the question of appendicitis. The diagnosis may be made by the discovery of tubercle bacilli in the stools, provided that the patient does not swallow his sputum.

Treatment.—This is purely symptomatic so far as the bowel is concerned.

Chronic Ischio-rectal Abscess is often due to tubercle. If it bursts spontaneously and leaves a fistula, the walls of the latter may be infiltrated by tubercle. Both abscess and fistula should be treated by incision and scraping.

Tuberculosis of the *liver* is not of clinical importance. Miliary tubercles of microscopic size are common. In rare cases, multiple abscesses with bile-stained contents have been found as a result of tuberculosis of the finer bile-ducts.

vi. Tuberculosis of the Genito-urinary System.

Tuberculosis of the Kidney, as part of an acute or chronic general infection, is of no clinical importance. The tubercles may be scattered irregularly all over the organ, or may be confined to the territory of a single branch-artery.

In many cases, however, tuberculosis of the kidney is an apparently primary disease (*renal phthisis*, *scrofulous kidney*, *nephro-phthisis*, *scrofulous* or *strumous pyelitis* or *pyelonephritis*), the virus being conveyed to the kidney by the blood. Sometimes it is due to extension, by way of the vas deferens, prostate, and bladder, or by way of the blood, from a tubercular testis. Occasionally it is manifestly secondary to tuberculosis of the lungs. Only one kidney suffers at first, but the other is often involved later on.

Males are more liable than females. The disease is most common in early adult life.

Morbid Anatomy.—The disease may begin in the cortex, and, as a result of caseation and softening, break into the

pelvis and infect the urinary tract. More commonly it begins in the pelvis, on the tips of the pyramids, and spreads thence both into the kidney and down the ureter. From the urinary tract the infection may travel along the genital tract to the testicle.[1] The kidney is thus gradually destroyed by the development of abscess-cavities whose walls are lined with caseous matter. The organ may maintain its normal size, or pyonephrosis may result from blocking of the ureter.

Symptoms.—These point to pyelitis, though the undue frequency of micturition and the pyuria may for a time suggest cystitis. Moreover, the bladder may itself be diseased. The urine contains albumen, pus, tubercle bacilli, and occasionally blood. It is generally acid, but is sometimes ammoniacal. Pain may not be troublesome, but renal colic may be induced by caseous matter passing down the ureter. There is tenderness in the region of the kidney, but seldom a large tumour. Hectic fever and emaciation are the principal general symptoms.

Diagnosis.—The diagnosis from calculous pyelitis depends on the history of the illness ; on the presence of tubercle elsewhere, and of tubercle bacilli in the urine ; and on the existence of a family tendency to tuberculosis.

Prognosis.—This is unfavourable, though life may be prolonged for many years, and cure occasionally takes place.

Treatment.—This is not satisfactory. If it could be ascertained that the disease is limited to one kidney, that organ should be excised. Nephrotomy and drainage may be justifiable if pyonephrosis is present. Otherwise treatment must be symptomatic.

Tuberculosis of the Ureter and Bladder is almost always secondary to disease of the kidney or prostate. The *ureter* is thickened by tubercular infiltration of its wall. The lumen may become blocked either by this thickening, or by pus, blood, or débris coming from higher up the urinary tract. Such blocking may give rise to renal colic.

[1] Simultaneous tuberculosis of the urinary and generative systems of the female (apart from acute miliary tuberculosis) is extremely uncommon.

In the *bladder*, tubercular infiltration is specially marked at the trigone, and leads to ulceration, which extends in area and in depth. The symptoms are those of obstinate cystitis, but with little or no pain. Disease may be detected in the prostate, vesiculæ seminales, or elsewhere, and the bacillus may be found in the urine. The prognosis is unfavourable, though the patient may survive for a long time. General treatment is the most important.

In Tuberculosis of the Testicle, the infection is generally brought by the blood (sometimes from tubercular lungs or lymph glands), though occasionally it is propagated from the urinary tract along the vas deferens. It is generally unilateral, and affects both children and adults. The disease begins in the epididymis, and spreads to the body of the testis and along the vas deferens (which becomes thickened) to the urinary organs. Softening, suppuration, and sinus-formation ensue. The prognosis is generally grave on account of the presence of other tubercular lesions.

Tuberculosis of the Fallopian Tubes is the most important tubercular disease of the female generative system. It is occasionally primary, and sometimes due to extension from tubercular peritonitis, but it is generally secondary to similar disease of the lungs or other organs. The condition is usually bilateral. There is great thickening of the tubes from tubercular infiltration of their walls, which are lined internally by caseous material.

Tuberculosis of the *uterus*, *ovary*, and *mamma* is so rare as not to require description here.

vii. Tuberculosis of the Nervous System.

1. Tubercular meningitis has been considered in connection with acute miliary tuberculosis, of which it is in most cases a part (p. 108).

2. A chronic localised tubercular inflammation of the pia mater is described, but is rare apart from underlying tumour.

3. *Tubercular tumour of the brain* (*scrofulous tumour*, *solitary tubercle*) is very common, and, setting aside syphilitic growths, constitutes more than half the total of all kinds of

cerebral tumour. It is generally found in the cerebral substance, without obvious connection with the membranes. This tumour is most common in the cerebellum and cerebral hemispheres, and is often multiple. It is most frequent in early life, and the personal or family history is often suggestive of tubercle. The growth occurs as a firm mass, of large or small size, and mostly of a cheesy aspect, but bounded by a gray, translucent zone of fresh tubercles. The symptoms it produces are those of brain tumour, and will be considered elsewhere. After syphilis, this is perhaps the most likely of cerebral tumours to be arrested in its growth.

viii. Tuberculosis of Serous Membranes.

Tuberculosis of the Pleura.—The opinion is now held that a large proportion, if not the majority, of the acute pleurisies formerly regarded as simple, idiopathic, and due to cold, are in reality tubercular. This view is founded to a large extent on the results of inoculation experiments, and on the subsequent histories of the patients. The condition is supposed to be an acute miliary tuberculosis of the pleura ; but post-mortem evidence is seldom obtainable, and cultures are often sterile, or the pneumococcus may be found to be present. In rare instances, the inflammation is suppurative.

Cases of *subacute* or *chronic* onset are also common, and may also, at times, become suppurative. Another chronic form is characterised, not by sero-fibrinous or purulent exudation, but by adhesion with great thickening. Hæmorrhagic pleurisies are often tubercular.

Secondary pleurisy is almost invariable in pulmonary tuberculosis. It may be simple or tubercular. (See under Diseases of the Pleura.)

The pericardium suffers from tuberculosis less commonly than the pleura or peritoneum. The condition is generally secondary to disease of the glands in the thorax ; sometimes it is due to extension from a tubercular pleurisy. As in the case of pleurisy, so, too, with pericarditis, cases which appear clinically to be primary frequently turn out to be tubercular. Occasionally the disease is *acute*, with sero-fibrinous, san-

guineous, or purulent exudation, in which case tubercles may be discovered on removing the fibrin. In other cases, the process is *subacute* or *chronic*, and the pericardium is adherent and thickened, with caseous matter, and perhaps tubercles, embedded in the thickened membrane. The symptoms vary according to the local condition, and also according to the presence or absence of tubercle elsewhere.

The PERITONEUM is very often the seat of tuberculosis, and this disease used to be known as ' Tabes Mesenterica ' (Latin, *tabes ;* Greek, *phthisis ;* English, *wasting ;* namely, general wasting of the body, associated with mesenteric disease). The old idea was that the diseased mesenteric glands intercepted the flow of chyle which ought to pass from the intestine through the glands to the thoracic duct and blood-stream, with the result that the patient starved. As a matter of fact, however, it is seldom that cases occur in which the general wasting can safely be accounted for in this way, and practically, as Gairdner has pointed out, the clinical condition known as tabes mesenterica is almost invariably tubercular peritonitis.

Etiology.—The disease is specially common in childhood (' consumption of the bowels '). In England and Wales, it causes five or six times as many deaths in the first five years of life as at all other ages put together. It is in some cases primary, in others secondary. Though tubercles are present on the serous aspect of a tubercular ulcer of the intestine, under the peritoneum, such an ulcer seldom gives rise to tubercular peritonitis. The disease is occasionally secondary to tuberculosis of the vas deferens. The Fallopian tubes are sometimes found affected, but it is not clear whether the peritoneal or the tubal disease is most often primary. Either may be secondary to the other.

Morbid Anatomy.—The disease may occur acutely, with sero-fibrinous or purulent exudation ; or it may be chronic, and characterised by either caseous or fibroid transformation. In the chronic caseous form, the serous surfaces adhere to one another, so that the loops of bowel are matted together. Caseous matter accumulates in the midst of the adhesions, and the omentum is thickened. There may be some sero-

fibrinous, or even purulent, exudation. A few tubercles are often present in the glands, but this is seldom a prominent feature. In the fibroid form, there is little or no exudation, the tubercles are hard, and the abdominal cavity may be obliterated by adhesions. The pleura is often tubercular.

Symptoms and Physical Signs.—The symptoms may be acute or chronic, or there may be no symptoms. As ordinarily seen in the child, the disease is chronic. There is enlargement, hardness, and often tenderness of the abdomen, with emaciation of the limbs. Liquid effusion is usually slight or absent. Light percussion yields a dull note over more or less of the abdomen, instead of the normal tympanitic note, while stronger percussion often yields a tympanitic note, showing that an increased thickness of solid material (thickened omentum, peritoneum, etc.) is in front of the air-filled viscera. Diarrhœa is common, and there is progressive loss of flesh and strength, with intermittent or remittent pyrexia.

Prognosis.—The great majority of cases admitted to the medical wards do well. Under suitable treatment, the disease ceases to progress; the diarrhœa is arrested; the fever gradually abates, and, after weeks or months, ceases; abdominal tenderness passes away; and the child puts on weight, and is cured so far as any tubercular process can be said to be cured, short of removal of the tubercular focus. This may happen though there is also disease of the pleura. A few cases end fatally by exhaustion from diarrhœa, etc.

Treatment.—This includes rest in bed; suitable diet; arrest of the diarrhœa by lead and opium or other astringents; a general remedy, such as creosote in emulsion, or carbonate of guaiacol; and a local application to the abdomen. For the last, iodoform ointment is well suited, or cod-liver oil may be rubbed in. After the diarrhœa has ceased, cod-liver oil and syrup of the iodide of iron should be given; and when all symptoms have disappeared, the child should be sent to the country.

Tubercular polyorrhomenitis, tubercular polyserositis, or *multiple serous membrane tuberculosis,* involves the peritoneum and pleura, or pleuræ, with or without the peri-

cardium. It may be acute or chronic, and when chronic may be either caseous or fibroid. The viscera may be free from tubercle in such cases.

ix. Tuberculosis of Lymphatic Glands (Struma. Scrofula).[1]

Tuberculosis of lymph glands sets in most commonly in early life, and as maturity is approached or attained, the disease in many cases ceases to make progress. As with phthisis, so with this disease there may be a very marked family tendency, even among those in excellent social circumstances.

Lymph glands (cervical, bronchial, or mesenteric) may become tubercular without recognisable disease of the mucous membrane or other tissue with which they are related, but there is little doubt that an unhealthy state of that tissue—*e.g.*, chronic catarrh of a mucous surface—diminishes the power of resisting the attack of tubercle bacilli. The infection may spread from gland to gland, and several enlarged and caseous glands may be welded into a mass by extension of the disease through their capsules. The cervical glands often soften and give rise to a chronic abscess, which bursts through the skin and may infect it. But even when superficially situated, caseous matter may long remain quiescent, and may become practically obsolete. Caseous mesenteric and bronchial glands often undergo calcification.

Tuberculosis of the *cervical* glands may be associated with catarrhal conditions of the mucous membranes, or with eczema of the skin of the head. The disease may spread to the supraclavicular and axillary glands.

The *bronchial* glands are very commonly affected, both in children and in adults. Thus, in adults they are often affected secondarily in ordinary phthisis. But the bronchial glands in children appear to be as susceptible to tubercle as

[1] From Latin *scrofa*, a sow, because swine are liable to the same condition. Scrofula used also to be called the 'king's evil' and the 'cruells' (French, *écrouelles*). See Scott, 'St. Ronan's Well,' chap. ii.

the lungs in adults, and the glands are frequently caseous in children when the lungs are perfectly healthy. Carr states that fully one-third of the post-mortem examinations in the children's hospitals of London reveal tuberculosis somewhere ; and he finds caseation of the bronchial glands in at least 80 per cent. of the autopsies on tubercular children.[1] The bacilli may reach the glands through the pulmonary alveoli without necessarily injuring the latter ; or by way of the intestine, lymphatics of the diaphragm, and posterior mediastinal glands ; or by way of the tonsils, cervical glands, and tracheal glands ; or possibly by way of the blood-stream. The disease in the glands may spread to neighbouring structures, such as lung, pleura, or pericardium. If the glands soften, they may burst into the trachea, bronchial tubes, or other organs. In young children, pulmonary tuberculosis is very often due to direct extension to the lung from caseous bronchial glands, and in them acute general tuberculosis is usually due to infection from the bronchial glands.

When tuberculosis of the bronchial glands is in active progress there is hectic fever with emaciation. Characteristic symptoms and physical signs are not likely to be produced, unless the glandular enlargement is very great. The former include cough, pain in the chest, dyspnœa, and stridor. Among the latter are distension of veins over the upper part of the chest, percussion-dulness over the manubrium, bronchial breathing over the dull area, unilateral enfeeblement of the respiratory murmur, and a venous hum over the manubrium when the head is thrown back—due, it is supposed, to pressure on the left innominate vein.

The *mesenteric* glands very often become tubercular, especially in children. In many such cases there is no ulceration of the bowel. There are no distinctive symptoms, and it is seldom that the enlarged glands can be felt. As already explained, it was formerly supposed that the clinical condition known as tabes mesenterica corresponded ana-

[1] Workman finds that in Glasgow the mesenteric glands are tubercular even more frequently than the glands in the chest, and my own experience inclines me to accept this view as correct.

tomically to scrofulous disease of the mesenteric glands, but this is a mistake.

General tubercular lymphadenitis is occasionally met with, and is apt to be mistaken for Hodgkin's disease. Where practicable, a gland-mass may be excised under local anæsthesia for diagnostic purposes.

Treatment.—Good air, food, and clothing are essential. Cod-liver oil, syrup of the iodide of iron, guaiacol carbonate, and arsenic, are the most important internal remedies. In the case of superficial glands surgical measures are often requisite.

The *spleen* may be the seat of miliary tubercles or of larger caseous masses. The condition is not clinically important.

The *suprarenal capsules* are the seat of tuberculosis in Addison's disease, which is described elsewhere.

x. Tuberculosis of the Skin.

Opinions still differ as to the diseases which should be included under this heading. The following are the most important.

Lupus vulgaris (*noli-me-tangere*) may commence at any age, but in three-fourths of the cases it begins before twenty. It is more common in girls than in boys. It is more common among the humbler classes than among the wealthy. It has been supposed to exert a protective action against deep-seated tuberculosis, but this is doubtful.

The primary lesion is a soft, yellowish-brown, translucent nodule of the size of a pin's head, which is embedded in the deeper layer of the true skin. The multiplication of these nodules, with inflammatory infiltration of the intervening skin, gives rise to an irregular, slightly raised patch. A nodule looks like a little mass of apple-jelly, and consists of giant cells, epithelioid cells, and small round cells, with very scanty tubercle bacilli.

The patch may undergo spontaneous involution in the centre, and leave a firm scar (*lupus non-exedens*) ; but more commonly ulceration takes place (*lupus exedens*), and a sore is developed which becomes covered with greenish-black

crusts. Apart from ulceration, the patch is covered with scanty fine scales.

Lupus may attack any part of the skin or mucous membranes, but prefers the nose and neighbouring part of the cheek. It is seldom symmetrical. It may cause necrosis of cartilage, but (unlike syphilis and cancer) never erodes bone.

The disease is very chronic, often lasting for several decades. Destruction of cartilage and cicatricial contractions about the nose, eyelids, ears, and other parts, give rise to serious deformity.

Diagnosis.—The commencement in early life, the extremely slow progress, the ragged edges of the ulcer, and the complete escape of bone, distinguish lupus vulgaris from *syphilis*. The apple-jelly nodules, the ulceration, the non-symmetrical arrangement, and the early commencement, distinguish it from *lupus erythematosus*.

Prognosis.—This is favourable as regards life, but much patience is needed for success in treatment. Recurrence is common, and more or less disfigurement may be expected.

Treatment.—At the present time the best results are being obtained from the application of various kinds of electrical and light rays, and especially from the X-rays and Finsen's light treatment. Apart from such measures, an attempt may be made to destroy the diseased tissue by salicylic acid. This may be applied in the form of Unna's salicylic acid and creosote plaster-mull, or as an ointment to be rubbed in night and morning. The salicylic acid has a selective action, and destroys the unhealthy tissue. Another method, which, however, is extremely painful, is to thoroughly scrape the patch with solid nitrate of silver ; this also selects the diseased tissue. Excision is, of course, not suited for the face. Another procedure is to scrape with a sharp spoon, and then apply nitric acid for a few seconds, the acid being thereupon neutralised by solution of sodium bicarbonate. Considerable benefit may occasionally be obtained from the persevering use of tuberculin.

Cod-liver oil and sea air are useful adjuncts to local treatment.

SCROFULODERMA[1] may result from infection of the skin over a caseous lymphatic gland which has softened and burst; but it sometimes occurs independently, by the development of tubercular nodules (*tubercular gummata*) in the skin or subcutaneous tissue. Softening and very chronic ulceration ensue.

The eruption may be in a rough way symmetrical, and it does not, like lupus, specially favour the face or exhibit apple-jelly nodules. The surrounding skin often has a violet tint, and sometimes shows a striking development of warts.

There are practically no subjective symptoms. The patient will probably present other evidences of struma.

The treatment consists in cauterising, or, still better, scraping the seat of lesion. Cod-liver oil and sea air are the general measures. In spite of thorough scraping, the disease may show an obstinate tendency to recur.

The ANATOMICAL TUBERCLE (*Verruca necrogenica, post-mortem wart*) occurs in mortuary porters and in others who, while handling dead tissues which contain living tubercle bacilli, have the skin inoculated. The wart is seen chiefly about the fingers. If it tends to spread, it should be destroyed by salicylic acid, caustics, or the cautery.

LICHEN SCROFULOSORUM (*L. scrofulosus*) is a rather uncommon eruption, which is chiefly seen in children. Though the patient is seldom suffering at the time from tuberculosis of the lungs or other viscera, there is generally a family history pointing to this tendency, and not uncommonly the patient himself shows signs of scrofula. The elementary lesion is a folliculitis. The changes commence around the bloodvessels and in the connective tissue at the base of the hair follicles. The resulting papules are reddish at first, but subsequently become paler. They are often arranged in clusters. The sides of the abdomen are their favourite seat. The eruption may be scanty or profuse. The tubercle bacillus has been found in the papules. The disease has been termed 'miliary tuberculosis of the skin.'

There is little or no itching. The papules may persist for

[1] This is sometimes included under the term 'lupus.'

months and then disappear. Several crops may develop before the eruption finally ceases.

The diagnosis depends on the presence in a child of papules and pustules, arranged in circles or less regular groups ; and on the presence of tuberculosis elsewhere.

The prognosis is favourable.

The treatment consists in the local application and internal administration of cod-liver oil.

Lupus erythematosus is by some included among tubercular diseases of the skin.

19. LEPROSY

(Elephantiasis Græcorum).

Definition.—A chronic infectious disease due to a specific bacillus, and characterised by the development of granulomata in the skin and mucous membranes (tubercular leprosy), or in nerves (anæsthetic leprosy).

Etiology.—The *Bacillus lepræ*,[1] which was discovered by Hansen in 1871, resembles the bacillus of tubercle in many ways, including the property of retaining the fuchsin stain after treatment with a mineral acid. Until quite lately the microbe had not been cultivated outside the body, but Rost has recently claimed to have succeeded in the attempt by extracting all the chlorine salts from the culture media. He has, moreover, prepared ' leprolin ' from the cultures, and this has been used in the treatment of leprous patients with excellent results.[2]

Leprosy is rarely seen before the third year of life, even in a child borne and suckled by a leprous mother, and all the other evidence goes to show that, as a rule, very prolonged and close contact is necessary before it can be transmitted from person to person. Even this is not always sufficient. The bacilli are found in the discharges from leprous sores, and in the saliva, sputum, and nasal secretions, when lesions exist about the mouth and nose. They have also been found

[1] The term ' lepra ' is generally used as synonymous with leprosy, but Willan applied it to psoriasis.

[2] *Brit. Med. Jour.*, February 11, 1905, pp. 294-296.

in the blood, milk, and urine of patients. It is therefore possible that they enter the body in various ways. The disease has been spread by infected clothing. It is an ancient and widespread malady, though far less common in Europe than it was a few centuries ago.[1] Great Britain has long been free from cases of endogenous origin. Hutchinson denies that leprosy spreads by contagion, except in such rare instances as when an individual with a leprous sore hands food to another person. He holds that it is usually due to the consumption of unsound fish.

Morbid Anatomy.—Ulceration of the nasal septum can often be detected in early cases, and it has been supposed that this represents the seat of inoculation and the primary sore. The principal anatomical change in the disease is a development of granulation tissue, in the form either of a distinct nodule or of a more diffuse infiltration. The nodules are met with particularly in the skin ; while the mucous membranes, nerves, testicles, ovaries, liver, and spleen may be seats of infiltration. The round cells contain 'arge numbers of bacilli.

Symptoms.—The period of incubation is believed to vary from a few weeks to many years. In a large number of cases, the patient suffers, during months or years before the characteristic phenomena appear, from feverish attacks, weakness, and pains in various parts.

Thereupon successive eruptions of erythematous spots appear, the spots varying in number and size. Some are pigmented ; others are unduly free from pigment. At first they may be hyperæsthetic and transient, but later on they tend to be anæsthetic and permanent (*macular* leprosy).

At a later stage, the granulomata appear, either in the skin (*tubercular* or *nodular* leprosy), or in the nerves (*anæsthetic* leprosy), or in both (*mixed* leprosy). The expression 'nodular leprosy,' while convenient clinically, is scarcely ever accurate, since in this variety the nerves are also involved ; it is really mixed leprosy.

In the *tubercular* or *nodular* variety, crops of nodules

[1] King Robert the Bruce died of leprosy at Cardross Castle, on the Clyde, in 1329.

appear at varying intervals, each eruption being attended by febrile symptoms for a day or two. At first the nodules appear chiefly on exposed parts, and when they are numerous on the face they give it a 'leonine' aspect. In a similar manner, the corneæ and other parts of the eyes may suffer, and the mucous membranes of the mouth, nose, larynx, pharynx, etc., may be seriously damaged by infiltration and ulceration. Leprosy of the ovaries may be associated with irregularity or absence of the menses.

In the *anæsthetic* variety ('nerve leprosy'), the superficial nerves can be felt to be thickened. There are symptoms, first, of nerve irritation (neuralgia, tingling, etc.), and, later, of nerve destruction (anæsthesia, muscular atrophy, bullæ, ulceration, and gangrene).

Diagnosis.—The nodules, the early anæsthesia, the maculæ, the non-suppurative enlargement of the related lymph glands in both varieties of the disease, the thickening of nerves, and, above all, the discovery of the bacillus in a nodule or anæsthetic patch, make the diagnosis certain.

Prognosis.—Anæsthetic is more chronic than nodular leprosy. When the latter has been diagnosed, the expectation of life is eight or ten years; in the anæsthetic variety it is twice as long. The patient commonly dies from phthisis, amyloid disease, or some intercurrent acute affection. The anæsthetic variety generally ceases to progress after a time, though, of course, leaving mutilations. But even a severe tubercular case may recover, so that the individual regains good health. Where recovery takes place, there is usually no tendency to relapse. Moreover, it seems certain that leprosy in an early stage may occasionally vanish and leave no trace of its former presence.

Treatment.—Lepers should be so far segregated, or at least supervised, as to reduce to a minimum the risk of spreading the disease, but without making the sufferers complete social outcasts. Surgical methods may be usefully employed in removing necrosed bone, stretching nerves for the relief of pain, etc. Wounds in lepers heal quite readily.

For the disease itself no specific cure is known, but of the

remedies which have yielded good results a few may be mentioned. Chaulmugra oil (oleum gynocardiæ) is perhaps the best ; it is given in milk or in capsules two or three times a day, and the daily allowance is gradually increased from 30 minims to 2 drachms. At the same time, the oil is thoroughly rubbed into the skin, either in the pure state, or saponified with lime-water, or diluted with olive oil. Gurjun balsam (wood oil, balsamum dipterocarpi) is used internally and externally in the same manner and dosage. Sodium salicylate and ichthyol have also been used internally ; while salicylic, pyrogallic, and chrysophanic acids have been employed for inunction. Calmette's antivenene (20 to 30 c.c. hypodermically) has occasionally yielded good results.

20. SYPHILIS (Lues Venerea. Lues. Pox).

Definition.—A specific infection of slow evolution, transmitted by inoculation or by inheritance. In the *acquired* disease, a lesion (chancre) develops at the seat of inoculation, and is followed by constitutional symptoms and affections of the skin and mucous membranes (secondaries) ; at a later date, by granulation-tissue growths in the viscera, blood-vessels, bones, and elsewhere (tertiaries), and sometimes by non-specific (parasyphilitic) degenerations of the nervous system. The *inherited* disease is characterised at an early stage by inflammation of the nasal mucous membrane, various skin eruptions, enlargement of the liver and spleen, and changes in the bones ; and at a later stage by lesions involving the teeth, eyes, ears, and joints.

Etiology.—There is good reason to believe that the specific organism of syphilis is the *Spirochæta pallida* described by Schaudinn and Hoffmann in 1905. It has been found in primary and secondary lesions, and in enlarged lymph glands in syphilis ; in blood obtained from the spleen by puncture on the day before the roseolar rash appeared ; and in the blood and organs of infants with congenital syphilis. The organism is an extremely delicate spiral, with pointed ends, and is actively motile. Its length

varies from 4 to 10 μ, and the number of its curves from six to fourteen.[1] Syphilis has been successfully inoculated in anthropoid apes, with the production of primary and secondary lesions, and the specific spirochæte has been found in these animals.

The *acquired* disease is usually inoculated on some part of the external genitals during sexual intercourse, but it may be transmitted[2] to the mouth by kissing, to the skin by tattooing or vaccination, to the nipple of a wet-nurse by a syphilitic child, or to the finger of the physician in midwifery practice (erratic or extragenital chancres). It may also be transmitted indirectly, as by feeding utensils. The *inherited* disease may be derived from the father alone (sperm inheritance), or from the mother alone (germ inheritance), or from both. The more recently the parent has acquired the disease, the more likely is the infant to be infected ; and, other things being equal, inheritance is more likely to take place if the mother is the sufferer, than if the father is at fault. In accordance with Colles's law, a woman who has borne a syphilitic child is herself immune, even though she shows no signs of the disease. If the mother becomes infected after conception, the child may or may not become syphilitic (placental transmission). Parents seldom transmit the disease after they have reached the tertiary stage.

i. Acquired Syphilis.

Incubation.—The incubation period varies greatly in different cases, but the average may be taken as five weeks.

Morbid Anatomy and Symptoms.—The *primary sore* is a granulation-tissue tumour (*hard*, *indurated*, or *Hunterian chancre*), which develops at the place of inoculation. It is situated in the true skin, and consists of a great accumulation of round cells. Endarteritis is seen in the vessels of

[1] A spirochæte has an undulating membrane and a flexible body, shows snake-like movements, and is regarded as belonging to the protozoa ; whereas a spirillum has terminal flagella and a stiff body, shows cork-screw movements, and belongs to the bacteria.

[2] *Syphilis insontium*, syphilis of the innocent.

the part. The chancre appears first as a red papule, which grows in extent until there results a sore with a flat or excavated top, and a well-defined, hard, elevated margin. The base feels like parchment if thin, and like cartilage or wood if of some thickness. A scanty secretion may ooze from the surface and dry into a scab. The related lymph glands undergo painless enlargement (*syphilitic bubo*). After a few months, the primary sore heals, leaving a scar.

The *secondary symptoms* set in about six weeks after the appearance of the primary sore. There may be slight elevation of temperature with malaise, but in rare cases there is high fever with daily intermissions. Cutaneous eruptions (*syphilides*) are common at this stage, and are of various kinds. In addition to their polymorphism, they are distinguished by their symmetry and superficial character, by the absence of itching, and by their tendency to give rise to copper-coloured pigmentation. The most common early rash is the roseolar or macular, which is generally best seen on the abdomen, and consists of red spots, at first effaced, but afterwards not effaced, by pressure. Other varieties are the papular, squamous, and pustular, the last of which is exceedingly like the eruption of small-pox. When the secondary eruption is well marked across the forehead, it is known as the *corona veneris*.

The fauces are often congested at this stage, and may present whitish, superficial, symmetrical ulcers like snail-tracks. Congested and slightly swollen areas, known as *mucous patches*, appear on the mucous membranes of the mouth, nose, vulva, and anus. Hypertrophy of the papillæ, especially in the mouth, and about the genitals and anus, gives rise to the *syphilitic wart* when the papillæ remain distinguishable, and to the *condyloma* when they are fused into one mass by over-growth of the interpapillary tissue.

Other symptoms observed in different cases are loss of the hair (alopecia), changes in the nails (onychia), anæmia, headache ; iritis, usually occurring from three to six months after the chancre, and generally attacking one eye shortly after the other ; still later, but rarely, retinitis and disseminated choroiditis ; pains in various bones, especially at night

('osteocopic pains'[1]); synovitis; and occasionally deafness. The liability to secondary symptoms usually passes off in the course of six or twelve months, but under proper treatment they may be got rid of in a few weeks.

The *tertiary symptoms* usually set in about the third or fourth year, but may appear much earlier or much later. They are commonly separated from the secondaries by a period of immunity, in which, however, some patients suffer from slight relapses ('reminders'). The tertiaries are non-symmetrical, and often single or but few in number. They tend to affect the viscera, bones, muscles, and bloodvessels, and to cause deep lesions of the skin and mucous membranes. They tend to spread locally, and a large proportion of them originate as specific granulation-tissue tumours, known as gummata. Tertiary symptoms probably occur in less than one quarter of the cases of syphilis that are not properly treated, and there is reason to believe that suitable treatment will reduce the proportion to less than 3 per cent. The *gumma* is a gray, translucent, or gummy-looking mass of tissue, which readily tends to undergo caseous necrosis at various points. It may develop in almost any tissue. If it is superficial, it is apt to cause ulceration, but if it is deeply placed, it tends to be partly absorbed and partly converted into fibrous tissue, in the midst of which some caseous matter may remain for an indefinite period.

Among the important tertiaries are deep ulcerations of the *skin*. In neglected cases, the discharges from these may dry and accumulate into limpet-shaped crusts (rupia[2]).

Periostitis may give rise to nodes on *bones*, and, if neglected, to necrosis.

Syphilis of the Circulatory System.—Gummata occasionally develop in the *myocardium*, and may give rise to aneurysm of the heart. They may also cause angina, syncope, and sudden death. Fibrous transformation of the myocardium may result from gummata, or from syphilitic disease of the

[1] ὀστέον, bone; κόπος, weariness.

[2] Hutchinson, however, regards rupia as rather a late secondary symptom.

coronary arteries. *Chronic aortic valvular disease* may be due to syphilis.

The *cerebral arteries* may be the seat of an obliterative endarteritis. The overgrowth takes place in the subendothelial tissue, and may block the lumen. The round-celled infiltration may extend to the middle and outer coats. Independently of this, gummata may develop in the adventitia.

Syphilis is also an important factor in the etiology of *aneurysm*, probably through the agency of endarteritis obliterans involving the vasa vasorum, and thus giving rise to atheroma in young syphilitic subjects.

Syphilis of the Respiratory System.—The *nose* may be the seat of important changes which sometimes cause terrible disfigurement. Gummatous inflammation of the mucosa and periosteum may proceed to softening, and may thus give rise to necrosis of bone. This involves most commonly the vomer, nasal bones, and palatal processes of the superior maxillæ. The septal cartilage is often destroyed. The symptoms include a fœtid discharge, whose odour is recognised by the patient to be offensive, nasal obstruction, etc.

Syphilis of the *larynx* is often a very late manifestation of the disease. It is specially apt to attack the epiglottis and vocal cords. It may begin in the larynx, or may spread to the epiglottis from the fauces. Deep ulceration, extensive loss of tissue, perichondritis, necrosis of cartilage, thickening and cicatricial stenosis are among the results. Cicatrisation may lead to adhesion of the epiglottis to the pharynx, or to adhesion and immobility of the cords. The voice may be but little affected, but it is often rendered permanently hoarse, and may be lost altogether. Stenosis may give rise to serious respiratory embarrassment and stridor. The quality of the cough will be influenced by the character of the structural changes. Swallowing is sometimes impaired. The general absence of pain, even on swallowing, is one feature of diagnostic value in this disease ; and another is the good general nutrition, such as is not likely to be observed in cancer or phthisis. If the case comes under observation at an early stage, the prognosis is good ; but if there are signs

of perichondritis or stenosis, there is a risk of acute œdema or dangerous cicatricial stenosis. Similar changes to those just described are occasionally met with in the inherited disease.

In the *trachea*, syphilis occasionally gives rise to infiltration, ulceration, and cicatrisation. It is the usual cause of tracheal stenosis. The stricture generally involves some length of the trachea at its lower end, and sometimes extends into the bronchi. The symptoms include dyspnœa and inspiratory stridor. There are no distinctively laryngeal symptoms, there are no signs of a tumour near the air-passages, and there may be a history of syphilis, or evidences of that infection in another part of the body. The outlook is unfavourable unless the lesion is confined to the upper end of the trachea, and thus accessible to the surgeon.

Syphilis of the *lung* is rare in the acquired disease. Apart from the rare gummatous disease, it has been supposed that chronic interstitial pneumonia is occasionally due to syphilis.

The lung is more frequently involved in inherited syphilis, which causes the white pneumonia of stillborn children. The lung is enlarged, dense, and white. The condition may be unilateral or bilateral. The pleura is generally unaffected.

Syphilis of the Digestive System.—This is most commonly met with in the *rectum*, and chiefly in women. Gummata develop in the submucosa and gradually give rise to stricture just above the sphincter.

Gummata in the *liver* give rise to characteristic appearances. They may be single or multiple, and each caseous mass is surrounded by fibrous tissue which sends off strands in various directions. The arteries in the affected area are the seat of endarteritis obliterans. Much of the caseous matter may ultimately be absorbed. With or without passing through a stage of caseation, the syphilitic granulation tissue develops into cicatrices, which constitute lasting evidence of the infection. There is often a deep depression of the surface of the liver over the site of an old gumma.

Syphilitic disease of the liver may give rise to no symptoms, and when these are produced they vary much in character. There may be pain and tenderness in the hepatic

region from slight perihepatitis. Enlargement and irregularity of the liver may suggest malignant disease. A large, prominent gumma may simulate a cyst. In exceptional cases there is fever, jaundice, or ascites. Albuminuria may be present, owing to waxy disease of the kidneys.

Syphilis is an important cause of amyloid degeneration in the liver as well as in other organs. Disease of the liver is common in infants with inherited syphilis.

Gummata in the *kidney* are of no clinical importance.

Syphilitic orchitis, gummatous disease of the *testicle*, or syphilitic sarcocele, is characterised by a painless enlargement, which generally undergoes complete resolution, but may go on to suppuration or to fibroid atrophy.

Syphilis of the Nervous System.—The lesions that may occur in the nervous system are various and of great importance. They include:

(1) The gumma, with its tendency to caseous and also to fibrous transformation. This varies in size, and may be single or multiple. It generally takes origin in the pia mater, and tends to compress or invade the brain or cord.

(2) Gummatous meningitis, again either caseous or fibrous, and affecting either hard or soft membranes. Within the head, it usually takes the form of a localised lesion of the pia mater. In the spinal canal, it occurs as hypertrophic pachymeningitis, which may seriously damage both spinal cord and nerve-roots. The cranial nerves are often damaged by syphilitic meningitis or gummata.

(3) Arterial disease. This may cause arterial occlusion, with consequent softening of brain substance; or, by replacing the normal elastic vessel-wall by extensile fibrous tissue, may lead to aneurysm of the larger cerebral arteries.

A gumma gives rise to the general and local symptoms of a rapidly growing and usually of a superficially situated tumour. Chronic local meningitis may irritate or damage the cortex or cranial nerves, and give rise to corresponding symptoms. Arterial disease affects in most cases the middle cerebral artery; the paralysis is often of sudden onset, often without loss of consciousness, and often preceded for days or weeks by severe headache. Some severe cases in which

there is a combination of endarteritis, gummata, and meningitis, simulate very closely general paralysis of the insane.

Amyloid degeneration is a well-known late consequence of the syphilitic infection.

Parasyphilitic, *Meta-syphilitic*, or *Post-syphilitic* affections are diseases to which syphilitic subjects are liable, but in which the lesions are not specific or peculiar to syphilis. They occur at times in non-syphilitic subjects, and are not amenable to antisyphilitic treatment ; yet they are so often a sequel of syphilis that this infection must be considered to have a causal connection with them. Moreover, they sometimes develop in a syphilitic individual before the tertiary phenomena are at an end, so that it may be necessary to give antisyphilitic treatment to a patient with parasyphilitic symptoms. Among the diseases belonging to this parasyphilitic group are locomotor ataxy, general paralysis of the insane, and ophthalmoplegia externa and interna. The relation of aneurysm to syphilis has been already mentioned (p. 147).

Diagnosis.—There is often no doubt as to the present or past existence of syphilitic lesions. In many cases, however, there is no clear history, and it is very likely that the symptoms were overlooked. It is common for a patient to deny syphilis while admitting gonorrhœa or admitting exposure to the risk of infection ; and where such exposure is admitted, we must often give him the benefit of the doubt by treating him with antisyphilitic remedies. At the same time, the skin, mucous membranes, and organs, including the special sense organs, must be carefully examined for any trace of present or past disease. Among the important points in the history are a sore on the genitals, sore throat, skin eruptions, loss of the hair, iritis, and, in the case of women, miscarriages.

Prognosis.—Much depends on the way in which treatment is carried out. If the patient is young, and the disease is treated thoroughly from the very commencement, and for a sufficient length of time after the symptoms have disappeared, syphilis is not likely to shorten life to any appreciable

extent. Treatment, however, is very commonly not of this thorough kind, and death is only too familiar an occurrence as a result of tertiary syphilis. In addition to this, temporary or permanent disablement from hemiplegia and other results of tertiary lesions is common ; while, of the parasyphilitic affections, locomotor ataxy, and still more general paralysis of the insane, are of grave importance.

A second attack of syphilis (with primary and secondary lesions) sometimes occurs, but is exceptional, and the second attack is usually modified.

Treatment.—Mercury is the remedy for the primary and secondary stages, and iodide of potassium for the tertiary stage. Mercury may be given in various ways: by the mouth, by inunction, by fumigation, or by intramuscular injection. One of the best modes is by a pill, containing 1 grain of gray powder (with or without 1 grain of Dover's powder to prevent diarrhœa), to be taken four times, or oftener, each day. As a rule, this pill should be continued for a couple of years after the disappearance of the secondaries. Or the solution of the perchloride of mercury may be given in mixture with iodide of potassium. If inunction is desirable, 1 drachm of freshly-prepared 10 per cent. mercuric oleate should be rubbed in twice a day (the same piece of flannel being used for all the applications), till the mouth is slightly affected, after which a smaller quantity should be used. The patient should live on the simplest fare. He should use a mouth-wash containing chlorate of potassium, and ought to keep his teeth thoroughly clean.

Iodide of potassium is conveniently given in solution (10 to 20 grains thrice daily). The course of iodide should not extend beyond two months, but it should be repeated several times in the first year, and twice a year for several years afterwards.

In the later stages, iron and other tonics and a change of air may be desirable. The patient should not marry until he has been at least two years free from symptoms, and he should be warned as to the contagiousness of the primary and secondary lesions.

ii. Congenital Syphilis.

Congenital or inherited syphilis appears to be exactly the same thing, whether derived from the mother, or derived from the father, or due to placental transmission. In the congenital disease there is no primary sore. It is rarely, indeed, that a child is born alive with signs of syphilis. When the fœtus suffers *in utero*, it is almost always killed by the syphilitic poison, a fact which may account for the fœtal deaths and the miscarriages so common with syphilitic women, though it is quite possible that the placenta, oftener than the fœtus, is at fault. The usual rule is that the fœtus remains healthy throughout intra-uterine life, and is born with all the appearances of good health.

After three or four weeks, nasal catarrh, with impeded breathing and 'snuffles,' sets in, and some skin eruption and fretfulness soon follow. If the rhinitis is very severe, it may lead to necrosis, so that the bridge of the nose is permanently depressed. The disease, unless treated, is at its height from the second to the fourth month, and the symptoms, apart from the snuffles, are very similar to the secondaries of the acquired disease ; but the general health suffers much more, and death not uncommonly results from the inherited disease. The most common form of eruption is a yellowish-red erythema, which begins about the buttocks and genitals, and tends to spread over a great part of the body. Sometimes there are fissures ('rhagades') about the lips or angles of the mouth, and these may leave permanent scars. As in the acquired disease, there may be not only a polymorphous rash, but also alopecia, iritis, mucous patches and condylomata, rheumatoid pains, etc., all symmetrical, transitory (whether treated or not), and readily amenable to treatment by mercury. There is often enlargement (simple hyperplasia) of the spleen ; less frequently enlargement of the liver (from diffuse interstitial hepatitis or from gummata) ; sometimes thickening of the bones around the anterior fontanelle, craniotabes (not peculiar, however, to syphilis), and epiphysitis. Disease of the liver in syphilitic infants is occasionally associated with gummata or cicatrices,

as in tertiary syphilis of the adult ; but usually the condition is a diffuse infiltration of round cells, such as might be expected in secondary syphilis. The cells are present between the hepatic cells, and the latter may degenerate. The condition has been called pericellular, intercellular, or monocellular cirrhosis. The organ is enlarged and firm, and often shows small spots, which are found by the microscope to consist of round cells. Occasionally, at puberty or adolescence, if treatment has not been employed, a liver of this kind may become the seat of lesions like those in tertiary acquired syphilis. It has been suggested that multilobular cirrhosis of the liver in children may be a parasyphilitic affection resulting from inherited syphilis.

In the course of a year or less, if the child survives, the phenomena which have been mentioned disappear altogether, and for some years no active symptoms are present. Then, for a further period of years, the patient is liable to suffer from lesions of various kinds, and after these in their turn have died out, he usually remains through life free from further trouble, though possibly disabled in some way.

Among the later phenomena just referred to are changes in the permanent teeth, especially the upper central incisors (' Hutchinson's teeth '). These are too short and too narrow, and have a single cleft in the centre of their free edge. Between puberty and the twentieth year, there may be deafness involving both ears, but not attended with pain or discharge. About the same time, or earlier or later, there may be interstitial keratitis. This affects both eyes and produces the ' ground-glass ' corneæ, but tends to complete recovery. Chronic synovitis of large joints, especially the knees, and periostitis of long bones, which may give rise to large and numerous nodes, are other phenomena of this period.

Gummata and amyloid disease are both rare in congenital syphilis.

Diagnosis.—In infancy, this depends chiefly on the snuffles, the desquamating erythema of the buttocks, face, and other parts, the sores at the angles of the mouth, the condylomata, and the history of the parents. Later on, the principal signs are : deafness of rapid onset in both ears,

without pain or discharge ; symmetrical interstitial keratitis ; Hutchinson's teeth ; disseminated choroiditis ; multiple chronic nodes of the long bones ; a depressed nose ; scars radiating from the mouth, and an earthy complexion.

Prognosis.—Death frequently occurs among the children of the poor, but very seldom among the rich. Syphilitic pemphigus is almost always fatal, but this is rare ; it generally appears in the first week of life. The serious and often fatal tertiary lesions of acquired syphilis almost never occur in the inherited disease. In adolescence, the eyes or ears may be damaged.

There is no satisfactory evidence that a syphilitic infection is ever transmitted to the third generation.

Treatment.—Syphilitic infants must be carefully clothed and fed. Mercury is the specific remedy. It may be given by inunction or by the mouth. A powder containing $\frac{1}{6}$ of a grain of calomel or 1 grain of gray powder, with some sugar of milk, may be given night and morning. The mercury need not be continued after the symptoms disappear. Condylomata and cutaneous ulcers should be treated with a mercurial powder or ointment. The mother should be warned as to the contagious character of the sores and their secretions.

When the later lesions set in, small doses of gray powder with tartrate of iron or syrup of the iodide of iron may be administered.

21. GENERAL GONORRHŒAL INFECTION

(Gonorrhœal Rheumatism).

Besides spreading by continuity in the genito-urinary system, and by inoculation to the conjunctiva, the gonorrhœal infection may invade the body generally, chiefly from the urethra, but occasionally from the conjunctiva. The *Gonococcus* has been found in metastatic lesions arising in this way, and it has been found in the blood, but it is possible that in some cases the toxin alone is absorbed. The usual changes induced by such general infection are in joints and fibrous tissues, but occasionally there may be pericarditis,

endocarditis, pleurisy, myelitis, iritis, and sclerotitis (apart from local inoculation of the conjunctiva). In some of these cases the infection is mixed.

The arthritis occurs chiefly in males, and may set in at any stage of the urethritis, or after this has ceased. It may be multiple and symmetrical, but is often confined to one joint. The knee, ankle, and wrist are specially susceptible, but any joint may be involved. The joint is swollen, painful, and tender, and effusion is present around it as well as within it. The condition is apt to persist for weeks or months, and may lead to permanent stiffness and even ankylosis. Inflammation of the fasciæ and sheaths of tendons is also common.

The **diagnosis** depends upon the presence or history of a specific urethritis, the obstinate character of the arthritis, the frequent involvement of only one joint, the involvement of fasciæ and tendon-sheaths, the occasional association of ophthalmia with the arthritis, and the affection in some cases of joints not often attacked by ordinary rheumatism (sterno-clavicular, temporo-maxillary, sacro-iliac, or intervertebral).

Prognosis.—The arthritis is generally cured in the long-run, but sometimes the joint is permanently damaged. In the rare cases where important internal structures such as the endocardium or spinal cord are attacked, the outlook is very grave.

Treatment.—The urethritis, if still in existence, should be promptly treated. Salicylates cannot be relied upon, but iron, quinine, arsenic, potassium iodide, and guaiacol deserve a thorough trial, one after the other if need be. Morphine may be required at night for the pain, but the local application of heat is preferable. Counter-irritation by blisters or iodine may relieve the pain and favour absorption. If the effusion within the synovial cavity is considerable, firm elastic pressure may be tried. A layer of cotton-wool about a foot thick is wrapped round the joint. The limb is then bandaged from the extremity upwards, and when the bandage reaches the cotton-wool it is wound as tightly as is possible. This causes no discomfort, and has often an extraordinary effect in promoting absorption. Failing this,

the joint should be opened and irrigated. Adhesions may be broken down under an anæsthetic. For cardiac and other internal inflammations, the treatment is that of the particular lesion. Unfortunately, there is no method yet available of combating a general gonorrhœal infection.

22. SAPRÆMIA, SEPTICÆMIA, AND PYÆMIA

(Septic Diseases. Blood-Poisoning).

Three distinct conditions are recognised as coming under the designation of septic diseases—viz., sapræmia, septicæmia, and pyæmia.

In *Sapræmia* (*Toxæmia* or *Septic Intoxication*) the poisonous products of micro-organisms, but not the organisms themselves, are absorbed into the blood. The organisms are mainly pyogenic or putrefactive, and they may be present in wounds, ulcers, serous cavities, etc. In the strict sense of the word, tetanus is a sapræmia, because the microbe remains in the wound, and the symptoms are due to the toxins absorbed ; but the phenomena are so distinctive as to entitle the disease to separate recognition. The slighter degrees of sapræmia are illustrated by the fever observed, with or without other symptoms, in cases of suppurating wounds or pulmonary cavities, acute abscess, empyema, decomposition of retained placenta, etc. ; while the more deadly results of sapræmia are seen in acute peritonitis, or when gangrene of the lung undermines the pleura and causes acute septic pleurisy. These toxins belong to the group of sepsins or ptomaines, and some of them are produced by organisms which have no power of invading the body (saprophytes), though such toxins may diminish the resisting power of the tissues, and so facilitate an attack of parasitic microbes.

Symptoms.—In a severe case the temperature rises, with or without rigors, and the patient suffers from headache, thirst, and sometimes vomiting and diarrhœa. If no more poison is absorbed, recovery may speedily take place ; but if rapid absorption continues, the typhoid state may ensue, and lead to a fatal issue very speedily or after several days.

Treatment.—The treatment is obviously to remove the organisms where this is practicable, whether by scraping wounds, opening and irrigating cavities, applying germicides, or otherwise ; and in any case to endeavour to maintain the general condition by means of food, stimulants, tonics, and fresh air.

Septicæmia (*Septic Infection*) is a more serious condition, inasmuch as the organisms, as well as their products, invade the tissues and usually the blood. There is often an obvious source of infection, as in a punctured wound, the sore throat of scarlet fever, an intestinal ulcer, or the uterus after labour ; but occasionally no local lesion can be discovered during life, or, it may be, even after death (' cryptogenetic septicæmia '). The microbe is one or other of the pyogenic organisms. The morbid anatomy is in the main that of the febrile state, but in puerperal cases there is often peritonitis.

Symptoms.—These at first resemble those of sapræmia, and the patient tends to lapse into the typhoid state. The fever is remittent or intermittent. A yellow colour of the skin, cutaneous hæmorrhages, and a sweet odour of the breath, may be present. There may be obstinate diarrhœa.

Diagnosis.—In septicæmia, the prostration tends to be greater than in sapræmia, and it may be possible to detect the microbe in the blood. On the other hand, disinfection of the part by which the toxins enter the body may bring to an end an attack of sapræmia. But in other respects it is scarcely possible to distinguish between septicæmia and sapræmia clinically.

Prognosis.—In a severe case of septicæmia, this is very unfavourable, but recovery has been observed in a case where the streptococcus was present in the blood for six weeks.

Treatment.—This is the same as for sapræmia, with the addition that antistreptococcic serum should be employed in cases where the streptococcus can be determined to be the invading microbe. This serum is antibacterial, but not antitoxic. Numerous races of streptococci ought to be used in immunising the horses, so as to furnish a ' polyvalent ' serum. Thirty c.c. should be given at once in an

acute case, and repeated daily. Serum should also be injected into the primary focus if this is accessible.

Pyæmia is septicæmia with the addition that multiple abscesses develop throughout the body. The *Streptococcus*, the *Staphylococcus aureus*, and the *Pneumococcus* are among the pyogenic organisms which may give rise to it. The disease arises under the same kind of circumstances as septicæmia, and the explanation of the abscesses in pyæmia is that a septic phlebitis has occurred in the neighbourhood of the primary lesion. The thrombus which develops in this way is invaded by the organisms and softens, with the result that portions of it are carried away in the blood-stream, and cause embolism of capillary or other vessels. The bacilli thus arrested multiply and give rise to secondary ('metastatic' or 'embolic') abscesses.

When the primary lesion is in a part drained by the systemic veins, the abscesses develop chiefly in the lungs, but some microbes may get through the pulmonary vessels, and settle on the endocardium, or pass into the arteries. When the original lesion is in a part drained by the portal system of vessels, the secondary abscesses are chiefly in the liver.

The abscesses in acute cases are small. In more chronic cases they are larger, fewer, and not so much in internal organs as in joints, subcutaneous tissues, the eyeball, etc.

Symptoms.—Among the more characteristic symptoms of pyæmia, there is the occurrence at irregular intervals of severe rigors with a high temperature, followed by profuse sweating and a rapid fall of temperature. In acute cases, the other constitutional symptoms are severe, and lead on to the typhoid state. Erythematous and hæmorrhagic eruptions may appear on the skin. The joints and serous membranes often suppurate, and death may supervene in a week.

Diagnosis.—This may be difficult if no possible source of infection can be recognised. At an early stage, there may be local evidences of septic thrombosis. The irregular occurrence of severe rigors, and the sweating, yellow skin, sweet breath, and cutaneous eruptions, are very significant. Multiple superficial abscesses will be observed in some cases.

In others, the signs and symptoms of metastatic abscesses will often be present (cough, dyspnœa, and localised consolidations of the lungs ; effusions in the pleuræ and pericardium ; peritonitis ; pain in and enlargement of the spleen ; albuminuria and hæmaturia ; enlargement and tenderness of the liver, sometimes with jaundice ; suppuration of joints ; panophthalmitis, etc.).

Prognosis.—Acute cases die in a week or two. Chronic cases may ultimately die from exhaustion, or may recover with ankylosed joints, or a shrunken eyeball, or some other deformity.

Treatment.—The treatment includes the removal or disinfection, wherever practicable, of any part from which infection might spread ; and the maintenance of the general strength by food, tonics, and stimulants. In cases due to the streptococcus, the appropriate serum may be tried. For superficial abscesses, surgical measures are indicated.

23. ERYSIPELAS

(Ignis Sacer. St. Anthony's Fire. The Rose).

Definition.—An infective inflammation of skin or mucous membrane, associated with constitutional disturbance, and due to the *Streptococcus erysipelatis*, which is present in the lymphatics.

Etiology.—The *S. erysipelatis* is now regarded as identical with the *S. pyogenes*, which is found in cellulitis, pyæmia, and puerperal fever. It is present in chains in the lymphatics at the spreading margin ; and as the inflammation spreads, the microbes die out in the central portions.

The disease is conveyed by direct contact, by fomites, and by the air. It does not very readily attack persons of robust vigour, but is predisposed to by alcoholism, by chronic disease, and by the presence of any wound or sore. In the latter case the inflammation generally starts from the wound (*traumatic* erysipelas), but under other circumstances (*idiopathic* erysipelas) the face and scalp suffer most frequently. Many believe, however, that even where

no cutaneous lesion is discoverable a small one must have existed to give admission to the microbe.

Morbid Anatomy.—The affected skin is swollen, hard, and perhaps disfigured by bullæ, but the redness passes away after death. Other appearances are due to the fever, and others—*e.g.*, abscesses—may be present as a result of complications.

Incubation.—The incubation period is usually from three days to one week.

Symptoms.—The invasion is sudden, with shivering, headache, vomiting, and high fever. Albuminuria is usually present, and diarrhœa is common. The inflammation in non-traumatic cases generally begins at the junction of skin and mucous membrane—*e.g.*, at the mouth, eye, or nostril. Sometimes it extends into the mouth, and in exceptional instances it begins in the mucous membrane ; but, as a rule, it spreads along the skin. The latter is red, swollen, tender, and painful, and bullæ often develop upon it. Sometimes, however, the colour of the skin scarcely differs from the normal. The great swelling of the cheeks, eyelids, lips, ears, etc., even without the addition of bullæ, causes great disfigurement of the features. The disease may involve one or both sides of the face. The margin of the inflamed area is well defined and elevated. The inflammation may be subsiding at one part whilst spreading at another. In severe cases there is delirium, and in debilitated subjects the typhoid state may ensue. Death may be due to exhaustion or to complications. Defervescence, with cessation of the spread of the inflammation, occurs after a week or so, and in most cases takes place by crisis (Fig. 12). The fading of the rash is followed by desquamation, and in the case of the scalp sometimes by temporary loss of hair.

A chronic form of the disease (*erysipelas migrans*) may gradually pass from one part of the body to another, often with but slight general symptoms.

Complications and Sequels.—Extension to the larynx may cause dangerous interference with breathing. Cellulitis is common, and pneumonia, ulcerative endocarditis, and inflammations of serous membranes are occasionally met

with. Baldness has been meintoned as an occasional sequel. On the other hand, chronic skin disease and inoperable tumours may undergo improvement in consequence of

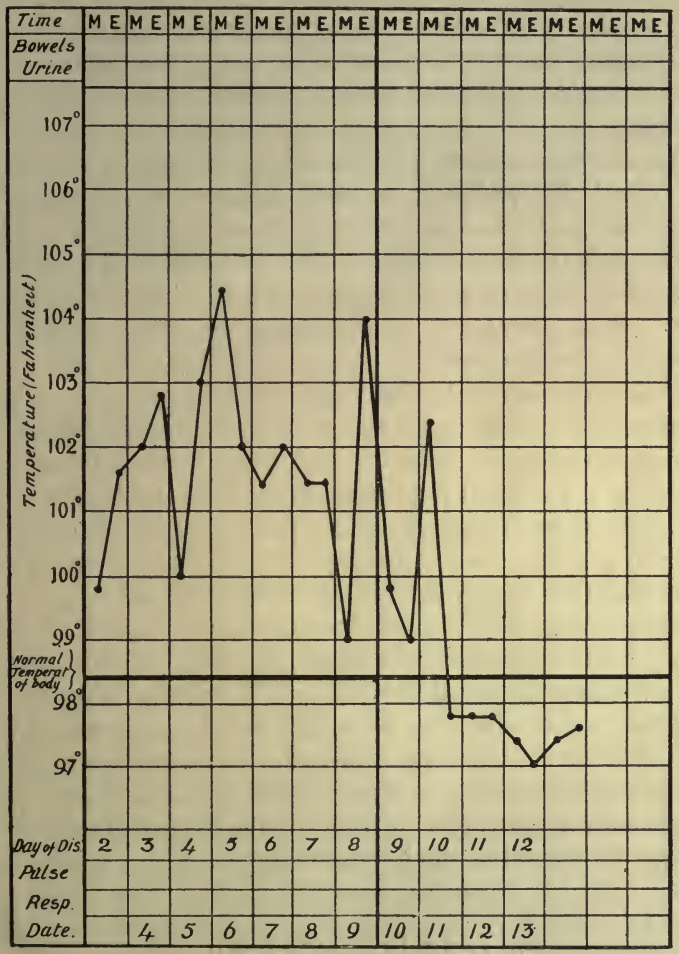

FIG. 12.—ERYSIPELAS OF FACE AND SCALP CONTRACTED BY A NURSE FROM A FATAL TRAUMATIC CASE.

an attack of erysipelas, and hence the introduction of Coley's fluid.[1]

[1] Coley's fluid contains the toxins of the *Bacillus prodigiosus* and the *Streptococcus erysipelatis* in combination, experiments having

Relapses are common, and some individuals suffer from many attacks of the disease.

Diagnosis.—This depends upon the severe localised inflammation of the skin, the well-defined, raised border of the inflamed area, and the severe constitutional symptoms.

In *erythema* there is no fever as a rule. The cutaneous redness around a *septic wound* or *abscess* is not sharply delimited.

Prognosis.—Conditions which strongly predispose to erysipelas (alcoholism, chronic visceral disease, etc.) influence the prognosis unfavourably. Erysipelas of the umbilicus in new-born infants is very fatal. Traumatic is rather more serious than idiopathic erysipelas. In the course of the attack, the prognosis will be influenced by the severity of the constitutional symptoms.

An attack confers little if any immunity.

Treatment.—This includes the general measures suited to all febrile maladies. In the old and feeble stimulants may be required. The most important drug is the perchloride of iron, which should be given in large doses. Antistreptococcic serum, which might be expected to act as a specific remedy, has thus far given very doubtful results.

To relieve the local pain, fomentations may be used (boracic acid, or belladonna with glycerin, or lead and opium). When fomentations are not employed, the skin should be dusted with zinc or starch powder, and covered lightly with cotton-wool to exclude the air. Sometimes the healthy skin close to the border of the diseased part is painted with solid silver nitrate, with a good result in arresting the spread of the inflammation.

24. TETANUS (Lockjaw).

Definition.—An infective disease due to a specific microbe, and characterised by persistent tonic spasm of the muscles,

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shown that the combined toxins are more strongly curative than the toxin of the *S. erysipelatis* alone. The organisms are grown in bouillon, and then killed by heat.

chiefly of the neck, face and trunk, often with paroxysmal exacerbations.

Etiology.—The bacillus, which was discovered by Nicolaier in 1894, is a slender rod with rounded ends, and develops in its interior, at one end, a spherical spore, which is so large that the spore-bearing bacillus resembles a pin or drumstick. The organism cannot grow in an atmosphere containing oxygen. It is plentiful in cultivated soil, in stable manure, and in the intestinal contents of many herbivorous animals. It never invades the blood or viscera, but remains localised to the seat of inoculation, and gives off its toxin. This is an intensely virulent poison which travels up the nerves, and can be obtained from the blood, urine, and viscera of the patient.

The disease is specially apt to follow severe lacerated and punctured wounds, compound fractures, and burns, but may result from the most trivial lesion. It is a well-known cause of death after injuries sustained in connection with the Fourth of July celebrations in the United States. The blank cartridge appears to be the principal agent in causing the infection in these cases. Experiments show that mere inoculation of the bacilli often fails to induce the disease. Some other condition is necessary, such as the presence of tetanus-toxin, of pyogenic organisms, or of traumatic damage to the tissues. Besides these *traumatic* cases, there are occasional *idiopathic* cases where no wound is recognisable ; these occur especially after severe exposure. It is possible, however, that in such cases the infection may take place through a crack in the lip, or hacks on the hands. In *tetanus neonatorum*, the 'eight-day sickness,' formerly prevalent in St. Kilda, the infection is by the umbilicus. *Puerperal tetanus* is frequent in hot countries. Tetanus has been transmitted by vaccination against small-pox and against plague, and also by the injection of diphtheria antitoxin. It has more than once broken out in hospitals as a result of surgical operations, probably owing to contamination of the catgut which is prepared from the intestine of sheep. Men suffer from tetanus more than women, owing no doubt to greater exposure to injury.

The dark races of man suffer more than Europeans, and the disease is far more common in hot than in temperate regions. In certain places the disease may be regarded as endemic.

Morbid Anatomy.—The wound in traumatic cases may be in any kind of condition. A distinct smile may still persist on the face at the autopsy. Occasionally a muscle is ruptured, or the spinous processes of the vertebræ are fractured, or the teeth are crushed down to their roots by the violence of the spasm. At times there is neuritis ascending from the wound, even as high as the spinal cord. Distension of vessels, ecchymoses, and round-cell infiltration have sometimes been observed in the central nervous system.

Incubation.—The period of incubation is usually from five to fourteen days, but may be shorter or longer. In one well-known case, in which a negro died a quarter of an hour after the injury was received, a quantity of toxin must have been introduced.

Symptoms.—The first symptom is often a feeling of stiff neck, or slight sore throat, or difficulty in opening the mouth (*trismus*, lockjaw). The eyes are partly closed, the forehead wrinkled, and the angles of the mouth drawn back so as to produce a smile or grin (*risus sardonicus*). These various phenomena are due to tonic spasm of the muscles concerned. The spasm increases in degree and extent. The abdominal muscles become rigid, and perhaps also the respiratory muscles and those which act on the proximal segments of the limbs. The distal parts of the limbs are but little affected, unless, it may be, near the seat of infection. Rigidity of the spinal muscles may cause backward arching of the spine (*opisthotonos*). Similar spasm of the abdominal muscles may cause *emprosthotonos*. As the spasm increases, it becomes painful, and, in addition, there are frequent paroxysmal exacerbations, which last from five to fifteen seconds each, and cause great suffering. They may be accompanied by profuse perspiration. The spasm generally ceases during sleep, whether this be spontaneous or induced by chloroform. Micturition is often difficult.

In very severe cases there may be considerable fever, but in others the pyrexia is accounted for by the associated sup-

puration, and in many instances the temperature is normal throughout. The pulse is accelerated and is often very small. Consciousness remains unaffected. In fatal cases death results from asphyxia, syncope, or exhaustion.

Varieties.—The principal variety that need be mentioned is *cephalic tetanus*, which results from injury to the head. In this form, the early trismus is accompanied by paralysis of the face on the side of injury, and often by tonic spasm on the other side; sometimes also by pharyngeal spasm (*hydrophobic tetanus*).

Diagnosis.—In *strychnine poisoning*, the convulsions are clonic and not tonic; they affect the whole body; they never begin with trismus; and they develop more quickly than those of idiopathic tetanus. In traumatic tetanus, the wound is an element in diagnosis, and the discharge may be examined bacteriologically.

In *hydrophobia*, the first spasms are respiratory, and are excited by attempts to swallow; there is no tonic rigidity, and there may be mental disorder. Cephalic tetanus may, like hydrophobia, be associated with respiratory spasm and with difficulty in swallowing, but in such cases a wound of the head and facial paralysis are present.

Hysterical trismus, or lockjaw, is complete in degree, and is not associated with stiffness in the neck; it may set in suddenly, last for hours or days, and then cease. Other hysterical phenomena may be present.

In *tetany*, the distal parts of the limbs suffer most, and their posture is sufficiently distinctive.

Prognosis.—Next to hydrophobia, tetanus is perhaps the most deadly of acute infections. The mortality of traumatic cases is about 90 per cent., and of the acute cases an even greater proportion die. About half of the idiopathic cases die. Tetanus neonatorum is as fatal as traumatic tetanus. From puerperal tetanus, recovery is extremely rare.

The duration of fatal cases of tetanus is generally less than two weeks; patients who survive that period have a fair chance of recovery. When the symptoms do not begin within ten days after the injury, there is a considerable chance of survival. In cases that recover, the disease always

passes off gradually, the spasms becoming less frequent and severe, and the tonic rigidity afterwards continuing slowly to diminish.

No sequels or second attacks are known.

Treatment.—A powerful antitoxic serum is available, but, unfortunately, thus far it has not yielded brilliant results. The explanation is that by the time symptoms of tetanus appear, the toxin has already entered into combination with the cells of the central nervous system, and in particular with the motor cells of the spinal cord. The dose is 20 c.c. of the serum injected in five different places (100 c.c. in all) at the same time, and this should be repeated on the two following days. Better results may perhaps be looked for from injection of the serum into the cerebral subdural space, or into the cerebral ventricle, or by lumbar puncture, the first of these three routes being perhaps the best.[1] In addition to this, in acute cases, or, without touching the brain, in the more chronic cases, subcutaneous injections may be given to neutralise any toxin that may be circulating in the blood.

Chloroform may be necessary to remove a severe spasm, so as to prevent asphyxia or permit of feeding. When asphyxia threatens in course of a spasm, artificial respiration ought to be employed. Chloral (2 to 4 drachms or more, daily, for an adult), long continued, is the best drug. As the case recovers, bromide may gradually replace the chloral. Morphine is another useful remedy, and may be used to procure sleep, while chloral is administered more continuously; or it may be given by itself at short intervals over a long period. The one other drug that need be mentioned here is Calabar bean; the extract of physostigma may be tried subcutaneously in doses of $\frac{1}{3}$ to 2 grains. It does not easily poison a patient with tetanus.

The wound should, of course, have thorough attention, and it may be desirable to give an anæsthetic and scrape or excise it. Moreover, in every instance where a wound has been received which appears to involve even a slight risk

[1] For intracerebral injection, it is best to use a specially powerful serum prepared without an antiseptic.

of tetanus, the wound should be kept thoroughly aseptic from the first, and should in addition be treated with dried antitetanic serum, while the patient should have a prophylactic hypodermic injection of 20 c.c. In veterinary practice prophylactic treatment has proved perfectly reliable.

25. GLANDULAR FEVER.

Definition.—An acute infectious fever characterised by multiple inflammation of the lymphatic glands, and especially of those in the neck.

Etiology.—The specific virus is unknown. The disease is almost confined to children, and tends to attack all the children of a household. The infection is supposed to enter through the throat or the intestine, though without causing any lesion of the mucous surface.

Incubation.—The usual incubation period is one week.

Symptoms.—The onset is sudden, with fever and sickness. The child seems to have a stiff neck, and complains of pain on swallowing. There is tenderness in the anterior triangle of the neck, usually on the left side, and on the second or third day the cervical glands beneath and in front of the sterno-mastoid muscle become obviously enlarged. Other superficial lymph glands may become involved, and the mesenteric glands, spleen, and liver may also be enlarged. The duration of the fever is commonly about a fortnight. The pain in the glands subsides quickly, the swelling slowly. The bowels are generally constipated during the attack, and afterwards loose.

Complications and Sequels.—Nephritis, otitis media, and suppuration of the inflamed glands are rare complications. General debility and diarrhœa may be troublesome sequels.

Diagnosis.—The occurrence of the disease in all the children of a household, and the absence of lesions of the mucous surfaces, are important points. If *plague* is epidemic at the time, a bacteriological examination should be made.

Prognosis.—This is favourable.

Treatment.—This is purely symptomatic. Fomentations to relieve severe pain, and tonics and change of air in convalescence may be recommended.

26. MEDITERRANEAN FEVER

(Malta, Rock, Neapolitan, or Undulant Fever).

Definition.—An endemic, and sometimes epidemic, infectious fever, due to a specific micrococcus, and characterised by a prolonged, indefinite, and irregular course, by splenic enlargement, usually by constipation, profuse sweatings and neuralgiæ, and often by arthritis.

Etiology.—The specific microbe is the *Micrococcus melitensis*, discovered by Bruce in 1886. It is constantly present in the spleen and other organs in fatal cases. It can be cultivated artificially, and is pathogenic in monkeys. It can live in urine for several days, in milk for weeks, and in dust and on dried fabrics for months. It is uncertain whether the organism is disseminated by air, by water, by mosquitoes, or by other means. The disease is not communicated directly from one person to another, but it has frequently been induced by accidental inoculation under the skin in the laboratory.

The disease is endemic on the shores of the Mediterranean and the Red Sea, and elsewhere. It is specially prevalent in the hot, dry season. It is most common between the ages of ten and thirty. Sex and social position have little or no influence.

Morbid Anatomy.—The appearances are in the main those of fever. The spleen is large and soft, and there is often pneumonia. There is no swelling or ulceration of Peyer's patches.

Incubation.—The incubation period is believed to be from three to ten days.

Symptoms.—These commence gradually, and in the early period include headache, insomnia, anorexia, vomiting in some cases, constipation, slight cough, and profuse perspiration. The spleen and liver are enlarged, and sometimes tender, and slight changes may be detected at the bases of the lungs. Sweat eruptions appear. Delirium is quite exceptional.

After a week or two, the headache and other symptoms of

acute illness pass off, the appetite is restored, and the tongue is clean, though the constipation continues. From this time the disease follows a very tedious course. There is progressive loss of flesh and strength, with fever, which is often high, and profuse sweating. The monotony of this long illness is frequently broken by complications, among which arthritis, neuralgia, and orchitis may be specially mentioned. The arthritis may, as in rheumatism, affect one or many joints, and may flit about from one joint to another.

The course of the temperature permits us to distinguish three clinical types of the disease. The most common is the *undulatory* type, in which the temperature over and over again rises gradually, as in enteric fever, and then falls to the normal. There may be half a dozen such periods of pyrexia. The first is generally the longest, lasting about three weeks ; the average of the series is about ten days. A second type is the *intermittent*, which is characterised by hectic fever, the daily fall of which is associated with profuse sweating. In the third, or *malignant* type, which is rare, the onset takes place suddenly with headache, the temperature rises high, the typhoid state supervenes, and death results.

Diagnosis.—The course of the fever, with its repeated relapses, the absence of localising signs (apart from the splenic enlargement and complications), the constipation, the absence of any eruption, and the endemic prevalence of the affection, are commonly sufficient for the diagnosis. But in many cases the resemblance to *enteric fever* is for a long time so close, that the agglutination test should be employed. The blood-serum from a case of Mediterranean fever acts upon cultures of the *Micrococcus melitensis* in the same way as the serum from an enteric case acts upon cultures of the bacillus of enteric. In the case of Malta fever, the reaction may be obtained as early as the second or third day, and with a dilution of 1 in 100 or more. The diagnosis from *malarial fever* can be made by examination of the blood.

Prognosis.—The mortality is only 2 per cent., but the average duration of the fever is nine or ten weeks.

An attack probably confers some protection, since a second attack is rare.

Treatment.—This is symptomatic, no specific remedy being known. The usual fever-diet must be given, but in the intermissions richer food should, if possible, be allowed, so as to sustain the strength and nutrition as much as possible, in view of the long duration of the disease. It is important that the patient should have flannel clothing next the skin, on account of the profuse sweating. Quinine, hurtful in the acute stage, may be given as a tonic later on ; and in convalescence the patient should reside in a country where he can spend almost all his time in the open air.

27. CHOLERA (Asiatic or Epidemic Cholera).

Definition.—An acute infectious disease, endemic in Lower Bengal, and thence often spreading as an epidemic to other countries ; associated with the presence of a specific microbe in the intestines and stools ; and characterised by vomiting, purging, muscular cramps, suppression of urine, and a high mortality.

Etiology.—The specific microbe is the comma bacillus (cholera vibrio, cholera spirillum), described by Koch in 1884, an actively motile, curved bacterium, whose length is only about a quarter of the diameter of a red corpuscle. It is furnished with a flagellum at one or both ends. It is constantly present in the intestinal contents and evacuations in cholera. It is most abundant at the height of the disease, and may then furnish an almost pure culture. It may pass through the epithelial layer of the intestinal mucous membrane, but it is not found in the blood or viscera.

Some individual predisposition is necessary before infection can take place, and this is probably often brought about by catarrh of the intestine—the cholera organisms multiplying in the albuminous exudate. The disease spreads only along the routes by which man travels, and it never travels faster than man. In most cases the disease is transmitted by water which has been contaminated by cholera

stools; the water may have been used for washing, for drinking, or for cooking. Milk, vegetables, and other foods may be contaminated. Possibly, in some instances flies may be the means of transmitting the infection. In a few cases the disease has been produced by accidental oral infection with laboratory cultures. Defective sanitary arrangements favour the spread of the disease. It does not pass directly from man to man.

The symptoms are due to the toxin produced by the microbe, which multiplies rapidly on and in the intestinal mucosa.

Morbid Anatomy.—Rigor mortis sets in early, and post-mortem contractions may cause movements of the corpse. The intestines contain material like the rice-water stools. The mucous membrane is sometimes pale, sometimes congested. The serous coat is injected. Peyer's patches and the solitary glands of the ileum are swollen and red. The spleen is usually small. The voluntary muscles are either dark or pale; an occasional one may be found ruptured as a result of the cramps during life. The kidneys are congested, and their epithelium may be cloudy or detached.

Incubation.—The incubation period is usually about three days (from a few hours to ten days).

Symptoms.—The period of symptoms is sometimes divided into four stages: (1) Premonitory stage; (2) evacuation stage; (3) collapse or algid stage; and (4) reaction stage.

(1) The first stage, when present, is characterised by the 'premonitory diarrhœa,' with which there may be nausea, discomfort at the epigastrium, and considerable prostration.

(2) Often, however, and especially at the commencement of the epidemic, when the disease is most deadly, the person appears to be in good health till he is suddenly seized with symptoms of the second stage. Stools of great volume are discharged in rapid succession, and without pain. At first the evacuations are fæcal in character, but they soon lose their bile-staining and come to resemble rice-water, with some flocculent matter suspended in it. The latter consists of epithelial and amœboid cells, with the specific microbe and other bacteria. Frequent vomiting also occurs, and, after a

time, the muscles of the abdomen and limbs become the seat of intensely painful cramps. Great thirst and restlessness are present. The patient soon passes into the third stage.

(3) In the third stage, the surface is cold, the eyes sunken, the urine scanty and albuminous or suppressed, the pulse scarcely perceptible, the voice lost, and the axillary temperature several degrees below normal, though the rectal temperature may indicate considerable fever. By this time the diarrhœa and pain may have ceased.

(4) If the patient survives this collapse stage, he may pass either into the tepid stage or into the stage of reaction. In the *tepid stage*, the temperature rises to about the normal, and the pulse may be felt at the wrist. Purging and vomiting cease, but the patient lies semi-comatose, with fixed pupils and hazy corneæ. This stage ends fatally. On the other hand, when *reaction* sets in, the surface may become warm, the voice and the functions of the kidneys may be restored, and recovery may result. But the typhoid state, suppression of urine, or some other complication of the reaction stage, may lead to a fatal issue.

Cases of cholera vary greatly in severity. Some are 'ambulatory'; others prove fatal in less than three hours. If the diarrhœa is severe, while the general symptoms are mild, the condition is sometimes distinguished as *cholerine;* it may pass on to recovery or to the fully developed disease. In *cholera sicca* the toxæmia is so intense that the patient dies before purging begins. The earlier cases in an epidemic of cholera are more severe than the later ones.

Complications and Sequels.—Among the complications are pneumonia, uræmia, and a cutaneous rash (*roseola cholerica*). Pregnant women usually abort, either during or soon after the attack. Among the sequels are chronic enteritis, anæmia, insomnia, ulceration of the cornea, gangrene, parotitis, and thrombosis in the right side of the heart or pulmonary artery.

Diagnosis.—A mild attack of cholera may be simulated by various conditions associated with severe diarrhœa, by food poisoning, and by malarial fever. Epidemic incidence is not peculiar to cholera, but in the course of an epidemic of

cholera, the rice-water stools, together with the symptoms described above, are nearly characteristic. The most conclusive evidence is the presence of the specific bacillus in abundance in the stools.

Prognosis.—The mortality is about 50 per cent.; it is higher than this at the beginning and lower towards the close of an epidemic. Childhood, advanced years, pregnancy, and chronic visceral disease, render the outlook less hopeful. In the course of an attack, the intensity of the symptoms and the rapidity with which the disease progresses are the basis of prognosis.

An attack of cholera confers immunity for a limited time. Thus, in the course of a single epidemic no one suffers twice, but an individual is in no way immune because he suffered in an earlier epidemic.

Treatment.—Haffkine recently introduced anticholeraic inoculation or vaccination as a preventive measure, and this has been largely practised in India with encouraging results. Immunity is conferred for a period of more than four hundred days. The individual is inoculated subcutaneously, first with a weak vaccine, and some days later with an exalted vaccine. The vaccines are emulsions made from attenuated and exalted cultures. It does not matter whether or not the living organisms are injected along with the cultures, since they die immediately after subcutaneous inoculation.

In presence of a cholera epidemic, the drinking-water, unless absolutely above suspicion, should be boiled, and milk also should be boiled before use. Diarrhœa should be promptly treated by appropriate diet and medicines. If it is the premonitory stage of cholera, this may not be of much avail, but it may well be diarrhœa of a much less formidable nature, which, nevertheless, if allowed to continue, will predispose strongly to a genuine choleraic attack. Aromatic sulphuric acid, chlorodyne, acetate of lead with opium, or Dover's powder, may be administered at short intervals to arrest such diarrhœa.

In the evacuation stage, absolute rest in bed in a warm room is essential. Ice or hot water may be given for the

thirst, and morphine may be injected subcutaneously for the cramps. A mustard plaster should be applied to the epigastrium to control the vomiting, and dry-cupping and poulticing in the lumbar region may be helpful if the urine is suppressed. No food should be given in this stage. Astringents are powerless to arrest the purgation, and purgatives which might be supposed to sweep the microbes and their toxins out of the intestine have been found by experience to be unsatisfactory. Intravenous injections of saline solution have occasionally been of distinct service, but it is better to introduce the solution under the skin.[1]

In the stage of reaction, the utmost caution is necessary in feeding the patient, and for a time only liquids can be given. These may be administered in teaspoonful doses every few minutes. Before long, raw meat-juice and beef jellies may be added. The convalescent should continue to take light meat foods for some time before starchy elements are added to his diet.

It is scarcely necessary to add that the evacuations, vomited matter, and fomites in a case of cholera must be thoroughly disinfected.

28. PLAGUE

(Pestis. Bubonic Pest. Bubonic or Oriental Plague).

Definition.—An acute infectious disease, occurring in epidemic form in various regions of the world, and supposed to be endemic in certain parts of India and China, associated with the presence of a specific microbe in the blood, tissues, and excreta, and characterised by fever, buboes, and a high mortality.

Etiology.—The bacillus was discovered by Kitasato and Yersin independently in 1894. It is easily obtained from

[1] *Normal* or *physiological saline solution* is a 0·9 per cent. solution of sodium chloride in water (say a drachm and a half in a pint). This is isotonic with the contents of human blood corpuscles. The fact that the earlier experiments were made on frogs' blood explains why a 0·6 per cent. solution was formerly employed. The fluid should be sterilised by boiling.

one of the diseased glands. It is also present in the blood, viscera, sputum, and excretions. It is a small rod with rounded ends. The ends stain more deeply than the middle, so that, when stained, the organism looks like a diplococcus.

The spread of plague is greatly favoured by defective sanitation. It rarely passes from person to person directly, and seldom through the medium of fomites, but it has been transmitted by inoculation of plague blood, by dissection wounds, and by accidental laboratory inoculation. Various kinds of animals are susceptible, including fleas, flies, dogs, mice, and especially rats. Rats are the principal transmitters of plague, and at the beginning of an epidemic they die in great numbers even before the people suffer. Their bodies show the characteristic lesions. Plague bacilli have been found in different parasites which live in the rat's fur, and it is probable that infected fleas of rats transmit the disease to man.

Morbid Anatomy.—Hæmorrhages are present in the serous membranes, and sometjmes in the mucous membranes and skin. The lymphatic glands are inflamed, and may contain foci of suppuration ; the infection spreads beyond their capsules, so that the surrounding tissues are bathed in serous, bloody, or purulent fluid. Hypostatic congestion of the lungs is usually present, and in a certain proportion of cases pneumonia. Enlargement of the spleen and liver, and congestion of the kidneys, are also recognisable.

Incubation.—The incubation period is usually from three to five days, and it occasionally happens that the patient feels out of sorts in the latter part of this stage.

Symptoms.—The invasion is sudden, and is characterised by great prostration, headache, and fever. The mental functions suffer early, and this is associated with a haggard, frightened, or perplexed aspect of the face, and sometimes with delirium. The temperature rises quickly to perhaps 104°, and the tongue becomes dry. The spleen is enlarged, the urine is scanty, and the typhoid state may supervene. Constipation is the rule, but sometimes there is diarrhœa.

In about three-fourths of the cases, buboes develop somewhere—most commonly in the inguinal glands, less fre-

quently in the axillary, cervical, or internal glands. These appear usually after several days, but occasionally at the very outset. The glands enlarge rapidly, and are often the seat of great pain. As a rule they suppurate (a favourable feature), in consequence of a mixed infection, and discharge foul pus and sloughs. If the patient survives, convalescence generally begins about a week after the onset, but it is often several months before it is completed.

Varieties.—The majority of cases belong to the *bubonic type*, which has just been described. *Pestis siderans*, or *septicæmic plague*, is a rapidly fatal type ; it may cause death within twenty-four hours. *P. minor*, *P. ambulans*, or *larval plague*, is so mild that the patient may not require to take to bed.

The *pneumonic type* of plague is particularly dangerous. It might easily be mistaken clinically for broncho-pneumonia, pneumonia, or severe bronchitis. There are no glandular enlargements, unless the pneumonia happens to be a complication of bubonic plague. Death usually takes place within five days. As the sputum swarms with plague bacilli, this variety is apt to infect those around the sick. It is even more deadly than the septicæmic variety, and appears to have prevailed in the ' Black Death ' (so called from the petechiæ, or ' plague-spots ').

Complications and Sequels.—Pneumonia and diarrhœa have been already mentioned. Pregnant women always miscarry. Convalescence may be disturbed or protracted through recurrence of the glandular disease, or by pneumonia, nephritis, abscesses, etc.

Diagnosis.—The most important points are the epidemic incidence, the sudden onset of fever, the special facies, and the painful buboes. The only evidence that is absolutely reliable is the presence of the *Bacillus pestis* in the buboes, blood, or secretions.

Prognosis.—About half of those attacked succumb, but in Eastern countries the mortality of the natives is sometimes much higher even than this. Death usually takes place within six days ; it may result from syncope, coma, asthenia, or pyæmia. A gradual onset, absence of severe symptoms,

and survival beyond the sixth day, are grounds for encouragement; but the temperature is not a satisfactory guide in prognosis.

In exceptional cases, a second attack has occurred.

Treatment.—Preventive measures include isolation of the sick, disinfection of houses, fomites, and excreta, destruction of rats, and great care on the part of attendants not to expose themselves to unnecessary risk, very specially if they have any wounds on their hands.

Haffkine's prophylactic vaccine, which contains the toxins from dead cultures, has been extensively employed in India, with encouraging results. Yersin's curative serum is obtained from horses which have been immunised by repeated inoculations. In the outbreak of plague in Glasgow in 1900, it was found that this serum had both prophylactic and curative value. While not affording complete protection, it appeared to modify considerably the severity of an attack. After the onset of an attack, it was almost useless when injected subcutaneously, but if introduced into a vein, it produced a marked beneficial effect. Failure was sometimes found to be accounted for by a mixed infection.

In other respects, the treatment must depend upon the symptoms that arise. Calomel may be given at the outset if there is constipation. Morphine is indicated for pain, insomnia, and diarrhœa. Sponging is a safe remedy for high fever, and cold applications to the head for delirium. If the heart shows signs of failure, stimulants such as ammonia, strychnine, digitalis, and bark are indicated. Glycerin and belladonna are commonly applied over the buboes, and when the time comes, these are to be incised, evacuated, and packed with iodoform gauze.

The patient should be isolated for a month after the acute symptoms have passed away.

29. YELLOW FEVER.

Definition.—An acute infection, endemic on the coasts of the West Indies and Central and Southern America, and sometimes appearing as an epidemic elsewhere; transmitted

by mosquitoes; and characterised by an acute febrile paroxysm, followed by jaundice, albuminuria, and a tendency to hæmorrhage, especially from the stomach.

Etiology.—The specific cause of yellow fever is not yet known, but in 1900 a commission of the United States Army demonstrated by experiments on soldiers in Cuba that the disease is transmitted by mosquitoes. The gnats were fed on yellow-fever patients, and after a fortnight were allowed to bite non-immune persons who had been kept rigidly in quarantine, with the result that these acquired the disease. The disease has also been transmitted to man by intravenous and by subcutaneous injection of blood from yellow-fever patients. It is not conveyed by fomites, and, as long as mosquitoes are excluded, there is no risk in nursing patients suffering from this affection.

The species of mosquito concerned in this disease is *Stegomyia fasciata*, the brindled or tiger mosquito. It bites in the daytime. Its larvæ are found in pots, tubs, tins, bottles, etc., where a little rain-water can collect, rather than in puddles on the ground.

The low-lying, insanitary areas of seaports are the favourite haunts of the disease; and warmth, damp, and deficiency of air and light tend to perpetuate it. The natives of yellow-fever areas are less susceptible than Europeans, but this is probably due to their having passed through an attack in childhood, and to the immunity thus induced being kept up by repeated inoculations.

Morbid Anatomy.—The principal post-mortem features are yellow staining of, and often hæmorrhage into, the skin and other tissues; in the early stage congestion, and in the later stages pallor, of the liver, with fatty degeneration of the hepatic cells; congestion of the kidneys, with fatty degeneration and desquamation of their epithelium. The stomach and intestine contain much altered blood. The spleen is normal.

Incubation.—The incubation period in the mosquito is ten days or more; in man, two to six days.

Symptoms.—The onset usually takes place suddenly with a rigor, prostration, and severe pain in the eyes, forehead,

back, and legs. The temperature rises to 103° or 104°, and remains at this level for several days, while the pulse-rate, which is rapid at the outset, gradually falls. The eyes are injected, the face looks bloated, and photophobia, thirst, anorexia, and epigastric tenderness are present. The patient occasionally dies in this 'primary fever.'

Preceding the second or hæmorrhagic stage, there has been described a 'period of calm'; but, according to Durham, it is seen only in books. In it the patient feels much better, but the pulse is abnormally slow, and albumen is present in the urine. After a few days more, definite convalescence may set in, or the second stage of the disease may ensue. This begins on the fourth day, and is characterised by remittent fever, intense prostration, in many cases delirium, deepening jaundice, severe albuminuria, scantiness or suppression of the urine, renewed pain in the epigastrium, and vomiting, at first of ingesta and then of material like coffee-grounds ('black vomit,' due to altered blood). Hæmorrhages take place from the mouth, nose, intestine, and uterus. Pregnant women miscarry. As the fæces may be normally coloured, and the urine free from bile, the jaundice must be largely toxæmic; but if jaundice is marked, the urine is bilious.

Varieties.—Some cases are mild, and even ambulatory. Others terminate fatally within a day or two. In occasional cases there is a relapse.

Complications and Sequels.—These include hepatitis, parotitis, abscesses, and diarrhœa.

Diagnosis.—There may be some uncertainty at first, chiefly as to the possibility of the attack being malarious, but the existence of an epidemic of yellow fever in the district, the absence of splenic enlargement, and the absence of the malarial parasite from the blood, will remove this doubt. The highly-flushed face, the slowing of the pulse while the temperature remains constant or rises, and the albuminuria, are three very characteristic early signs of the disease. In the course of a few days the absence of rash will exclude the eruptive fevers, and by this time the characteristic symptoms of the disease will be evident.

Prognosis.—The mortality varies enormously under different circumstances, and has been stated as ranging from 5 to 85 per cent. It has been estimated that fully two-thirds of all cases recover. In an individual case, chronic alcoholism, a high initial temperature, suppression of urine, delirium, black vomit, and the occurrence of a relapse, render the prognosis very unfavourable. Death generally occurs at about the fifth or sixth day, through suppression of urine, and possibly, in part, through the damaged liver permitting hurtful substances to pass from the intestine into the circulation. It rarely occurs after the tenth day.

A second attack is rare if the individual remains in the infected area, no doubt because he is constantly being re-inoculated. If, however, he goes to, and remains in, an uninfected country, he seems to lose his immunity after several years.

Treatment.—No specific is known. At the outset, the bowels should be cleared out by calomel and castor-oil, but later on enemata alone should be employed for this purpose. Fomentations should be applied to the loins for the early pains. If the urine becomes alarmingly scanty, dry cups and fomentations should be applied over the kidneys ; hot packs and large enemata of normal saline solution may be administered, and pilocarpine may be injected hypodermically. Phenacetin may be given for insomnia, but opiates must be avoided. Strychnine and digitalis may be indicated for a feeble circulation, and ice in small quantities may be used to allay obstinate vomiting. During the primary fever, it is better to give no food at all, but simply water in small quantities. In the calm and secondary febrile periods, only the lightest of liquid foods must be given in small, regulated doses.

Patients should be protected from mosquitoes by netting, and every effort should be made to destroy these insects, and to remove their breeding-places. There is good reason to hope that by exterminating the mosquitoes in any given area, yellow fever may in time be abolished. After it was proved that the mosquito was the transmitter of the disease, these means were adopted in Havana, with the result that yellow fever was completely abolished in the city.

30. WEIL'S DISEASE

(Infectious Jaundice. Epidemic Catarrhal Jaundice).[1]

Definition.—An acute infectious disease associated with enlargement of the spleen, icterus and nephritis.

Etiology.—A bacillus has been repeatedly found in the urine during life, and in the spleen, liver and kidneys after death, in cases of this disease. It seems to be allied to the *Protei*, bacilli which are present in decomposing organic matter ; and the history of some of the cases, and the fact that the disease is most common in the hot season of the year, are in accordance with the view that such an organism is the specific microbe.

Morbid Anatomy.—The cells of the liver are in a state of fatty degeneration, and many of them are quite destroyed. The kidneys are inflamed, and their epithelium is fatty or cloudy. The tissues are stained with bile. The spleen is sometimes enlarged, and the mucous membrane of the stomach and intestine may be inflamed.

Symptoms. — The definition given above includes the features recognised as characteristic by Weil, of Heidelberg, in his paper published in 1886. This writer described four cases in young adult males, but others have been recorded since then, especially in Germany. The onset is sudden, with febrile symptoms, including considerable pyrexia. Before long some or all of the following symptoms appear : head symptoms (headache, giddiness, delirium, and a tendency to stupor), jaundice, painful enlargement of the liver, enlargement of the spleen, digestive disturbance, and a condition of the urine pointing to acute nephritis. The fever continues for about a week, and defervescence takes place by lysis. Convalescence is slow, and is apt to be disturbed by a relapse. Weil's four cases all recovered, but some cases end otherwise. The mortality is probably between 10 and 20 per cent.

Diagnosis.—*Relapsing fever* is recognisable by the presence of spirilla in the blood.

[1] Described by Weil of Heidelberg in 1886.

In *yellow fever*, the spleen is not enlarged, and the pulse-rate declines after the invasion, while the temperature continues high for some days.

In *acute yellow atrophy of the liver*, that organ diminishes in size with a rapidity that can be recognised by percussion almost from day to day; the disease occurs chiefly in pregnant women, and commonly passes on to the typhoid state and a fatal issue, whereas Weil's disease generally ends in recovery.

Catarrhal jaundice is rarely met with in epidemics. Enlargement of the spleen is seldom well-marked. There is no nephritis, although an occasional tube-cast may be found in the urine. The abrupt onset with considerable fever, and the severe constitutional symptoms, observed in Weil's disease, are not met with in simple catarrhal jaundice. In the latter, indeed, apart from the jaundice, there may be no symptoms, except impairment of appetite and slight indigestion.

Treatment.—Antiseptics may be administered internally. Otherwise the treatment is purely symptomatic.

31. DENGUE

(Break-bone Fever. Dandy Fever).

Definition.—An acute specific fever of warm climates, characterised by severe pains in the joints and muscles, an initial erythematous and a later polymorphous eruption, and a very low mortality.

Etiology.—Graham, of Beyrouth, has brought forward evidence to show that the disease is due to a protozoon which is conveyed from the sick to the healthy by a mosquito, the *Culex fastigans*. Confirmatory evidence, however, is still required. The fever is very infectious, and will attack two-thirds or more of the population in a district where it is epidemic. No other disease can rival it in this respect. All classes of the community suffer alike. Outside the tropics, dengue occurs only in summer.

Morbid Anatomy.—As the disease is rarely fatal, few cases have been examined post-mortem. Softening of the cardiac

muscle, pericarditis, and effusions in joints have been observed.

Incubation.—The incubation period is one to five days.

Symptoms. — The invasion takes place suddenly, with shivering, high fever, and severe pains in the head, joints, and muscles. The joint-pains frequently commence in a finger. The face is bloated, the eyes injected, the pulse rapid, and the skin covered with an erythematous blush. Many joints may suffer together or in turn ; sometimes they are swollen. The red rash may be associated with a condition of throat and tongue suggestive of scarlet fever.

After three days or so, the initial fever comes to an end, by crisis or by lysis, and, apart from persistence of the pains or of stiffness, the patient feels pretty well. After two to four days of apyrexia, there is another paroxysm of fever, which is both slighter and shorter than the primary one, and is accompanied by the terminal exanthem, and by an increase in the pains. The rash is usually of a measly character, but may be papular, urticarial, or scarlatiniform. It spreads from the upper limbs to the face and trunk, and then to the lower limbs, and after a few days fades away in the same order. It is followed by branny desquamation. The second febrile period does not last more than a day or two, so that the total length of the disease is about eight days.

Relapses are not uncommon.

Varieties.—The disease may be very mild, but in rare cases collapse and fatal coma occur.

Complications and Sequels.—The peculiar pains may persist for a long time, and there may be considerable prostration. Among other sequels are boils, orchitis, and hæmorrhages from mucous surfaces.

Diagnosis.—The widely-spread epidemic incidence, the eruption, and the absence of sweating, distinguish the early febrile stage of dengue from *rheumatism*.

Scarlet fever is not an epidemic of the tropics, and is, moreover, distinguishable by the course of the temperature, and by the character of the eruption and desquamation.

Measles is not associated with severe pains.

In *influenza* there is no rash as a rule.

Prognosis.—The mortality is almost nil.

The immunity conferred by an attack is not complete.

Treatment.—The general treatment is that of the febrile state. The severe pains are to be relieved by phenacetin or phenazone, and, failing these, by belladonna or opiates. Iron and quinine should be given as tonics in convalescence, and if the pains persist, iodide of potassium may be of service.

32. BERI-BERI

(Kakké. Endemic Multiple Neuritis).

Definition.—A specific endemic and epidemic multiple neuritis, involving the vagus, phrenic, vascular, and other nerves; occurring chiefly in warm climates; attended by dropsy, and often ending fatally.

Etiology. — A micro-organism has been described by various observers, including Hamilton Wright, who finds a bacillus associated with the gastro-duodenitis of rapidly fatal cases; but this question is still unsettled. Wright holds that, whatever the specific organism may be, it enters the body by the mouth, and develops chiefly in the duodenum and pyloric end of the stomach. It there produces a toxin which is absorbed and poisons the peripheral nerves. He holds, further, that the organism escapes in the stools, and thus spreads the disease. Others maintain that the disease is not an infection, but a toxæmia, due to bad feeding, and rice is blamed by some, fish by others. The disease is common in Brazil, Japan, and the Malay Archipelago, but occurs in many other places. Though chiefly seen in warm climates, it has been met with in different parts of Europe, including Ireland, and occasional cases reach Glasgow by sea. It is not generally regarded as contagious, but it is apt to become endemic in certain localities or dwellings. Serious outbreaks have occurred in asylums in Dublin and the United States. Overcrowding greatly favours an attack. Young men suffer most frequently.

Morbid Anatomy.—In the wet variety of the disease, there is dropsy of the connective tissues and serous cavities; in the dry variety, the muscles are pale and wasted. The

heart is hypertrophied and dilated, and there is inflammatory degeneration of the peripheral nerves. In rapidly fatal cases hyperæmia and punctiform hæmorrhages are found in the duodenum and pyloric end of the stomach, and Wright looks upon this gastro-duodenitis as the primary lesion of the disease.

Symptoms.—The incubation period is said by Wright to vary from seven to twenty days. In severe cases the invasion is abrupt and characterised by symptoms which are often attributed to acute indigestion. The nervous phenomena set in within three days. In milder cases the onset is insidious, but among the early signs may be slight pitting over the shins, and changes in the electrical reactions of the muscles and nerves. Several varieties of the disease are described :

(1) In the *larval* or *rudimentary* form, there is impairment of movement and of sensibility in the lower limbs, with tenderness of the muscles, slight œdema, and perhaps some breathlessness and palpitation. After days, weeks, or months, these symptoms may pass off completely, or the attack may gravitate into one of the more serious types of the disease. If recovery takes place, the patient may have recurrences in warm weather.

(2) In the *atrophic*, *dry*, or *paralytic* form, there is rapid and extreme muscular wasting in the limbs, with loss of power, reaction of degeneration, and impaired sensation. The trunk generally escapes, but occasionally there is implication of the phrenic and vagus nerves. The patient may at any time die suddenly from cardiac failure, but in most cases recovery takes place after weeks or months.

(3) The *dropsical*, *wet*, or *hydropic* form is characterised by dropsy of the subcutaneous tissue and effusions into the serous cavities. Paralysis and anæsthesia are slight or absent. The urine is non-albuminous.

(4) Some writers recognise a *mixed* form, which combines the phenomena of the atrophic and dropsical.

(5) The *malignant*, *acute*, *pernicious*, or *cardiac* form is associated with grave symptoms of cardiac failure—dyspnœa, palpitation, dilatation of the right ventricle, vomiting,

etc. Death may occur within a few hours, but more commonly after some weeks.

Diagnosis.—Epidemic multiple neuritis in tropical countries is almost always beri-beri. An isolated case might be distinguished from alcoholic neuritis by the history.

Prognosis.—The mortality varies greatly under different circumstances ; perhaps about a fifth of those attacked die. The prognosis is grave if the patient cannot be removed from the place where he acquired the disease ; if severe cardiac symptoms are present ; or if great diminution in the quantity of urine is associated with serious dropsy.

Treatment.—Overcrowding in dwelling-places must be avoided. The patient should be removed to an unaffected locality, and should then rest in bed. The diet should be rich in nitrogenous constituents, and rice should be avoided. In New Caledonia, where beri-beri is common among all nationalities at the mines, water-cress is regarded as a cure, and is therefore largely cultivated by the mining companies. Purgation and bleeding will diminish the strain on the dilated heart. Nitrites should be freely used for dyspnœa, and strychnine should be given as a cardiac tonic. The salicylates have been recommended by one authority. Aspiration of the pleural cavities may be advisable.

33. VERRUGA (Verruga Peruviana).

Definition.—A specific infection, endemic in elevated defiles of the Andes, and characterised by fever, pains, and an eruption of granulomata.

Etiology.—A specific bacillus has been described as present in the interstitial tissue of the lesions. The disease is confined to certain valleys of the higher Andes, and attacks domestic animals, birds, and quadrupeds, as well as man.

Morbid Anatomy.—The cutaneous lesion or granuloma is said to resemble in structure a highly vascular sarcoma.

Incubation.—From one to six weeks is regarded as the usual incubation period. In the case of Carrion, a medical student who, in 1885, inoculated himself and died from the disease, it was twenty-three days.

Symptoms.—These vary greatly in different cases, but,

as a rule, the onset is gradual. Severe articular pains, intermittent fever, anæmia and weakness are prominent symptoms. The eruption appears after the lapse of from three weeks to many months, and at the same time the general symptoms undergo great improvement. The eruption affects first the skin of the limbs and face, but may spread over the rest of the surface, and may involve mucous and serous membranes. The small lesions are itchy red papules or warts, but larger ones may be as big as oranges. The growths gradually disappear, sometimes after ulcerating or bleeding so freely as to seriously aggravate the anæmic condition of the patient.

Diagnosis. — In some ways verruga is very like *yaws*, but the great tendency shown by the lesions to bleed is peculiar to the former. The diagnosis from some other febrile diseases may be very difficult until the eruption appears.

Prognosis.—The disease is attended by a considerable mortality, especially among those who are not natives of the district. An attack which is recovered from generally confers immunity.

Treatment.—The patient should be at once removed to the sea-coast, as the general symptoms and the tendency to hæmorrhage are then likely to become less serious. As malaria is often associated with this affection, quinine should be given freely.

34. YAWS (Frambœsia. Morula).

Definition.—A contagious, specific infection of tropical countries, which runs a chronic course, and is associated with an eruption of cutaneous granulomata.

Etiology.—The specific microbe has not yet been identified with certainty.[1] A breach of surface is necessary for

[1] Mr. Hutchinson considers the disease known as *yaws* in the West Indies and as *parangi* in Ceylon to be non-venereal syphilis (*New Syd. Soc. Atlas*, Fascic. xx., 1904.) Castellani has recently found in the lesions of yaws or parangi a spirochæte morphologically indistinguishable from Schaudinn's *Spirochæta pallida* (*Brit. Med. Jour.*, November 25, 1905, p. 1430).

inoculation, and the virus from a yaws sore may reach such a lesion either directly, or through the medium of clothing, insects, etc. The disease is thus specially apt to attack ill-clad children. Mothers, indeed, often inoculate their children intentionally. It is endemic in many tropical regions of both hemispheres.

Morbid Anatomy.—The cutaneous lesion involves the papillary layer of the cutis and the Malpighian layer of the epidermis. It consists of granulation tissue.

Symptoms.—The primary eruption ('primary sore') appears from fourteen to sixty days after infection. It may be preceded for some days by feverish symptoms and rheumatic pains. This initial papule develops at the seat of infection in inoculation cases, but is sometimes absent when the disease is acquired in the ordinary way. The generalised or 'secondary' eruption is usually delayed for several weeks after the primary sore. It begins in the form of itchy papules, each of which is at first about as large as a pin-head. The papule may abort at this stage, but more commonly it grows and bursts through the epidermis, on the surface of which it looks like a little red hemisphere, with some yellowish matter on the top. The yellow matter spreads over the lesion and dries into a crust. The lesion varies in size from a pea to a walnut, and after some weeks disappears, leaving a stain. The eruption may be scanty or abundant, and the 'yaws' generally appear in successive crops during a considerable period. The duration of the whole disease varies from a few months to a few years.

Diagnosis.—The cutaneous lesions just described, preceded by fever and pains, and occurring in a region where yaws is endemic, are quite characteristic.

Prognosis.—The patient recovers after months or years, and is usually immune thereafter.

Treatment.—Good food and hygiene are important. Iodide of potassium is the recognised remedy. The lesions may be treated with some local stimulant, such as chromic acid or sulphate of copper, or with an antiseptic ointment. The patient ought to be isolated, and the clothing, dressings, etc., should be disinfected.

35. ANTHRAX

(Malignant Pustule. Wool-sorter's Disease. Splenic Fever).

Definition.—An infection due to a specific bacillus, and attacking chiefly herbivorous animals, though sometimes acquired by man, in whom it may assume various forms.

Etiology.—The *Bacillus anthracis* is a long, non-motile rod with square or slightly concave ends, which under certain circumstances develops a very resistant spore in its interior.

The disease appears in sheep, cattle, horses, and other animals under various aspects, causing great enlargement of the spleen. Hence the name 'splenic fever,' which, however, is not so applicable in the case of man. It is seldom seen in animals in Britain, and among human beings it is almost confined to butchers, tanners, wool-sorters, hair-cleaners, and others who work with animals, hides, hair or wool imported from abroad. Cattle are infected by the swallowing of spores developed by bacilli which have escaped from diseased animals in the evacuations.

Symptoms and Morbid Anatomy.—In man the skin may be inoculated through a wound or scratch, and accordingly the initial lesion is generally on an exposed part, such as the face, neck, or hand. This is the *external* form. A papule appears here, and develops into a vesicle, which in turn dries into a black scab or eschar. A ring of small vesicles surrounds this dark area, and a firm œdematous swelling extends from it all round. This local lesion is the *malignant pustule*, but the fluid in it is serous, and not purulent. There is but little pain. At first the bacilli are limited to the neighbourhood of the pustule, and there is no great constitutional disturbance; but in the course of four days they are disseminated through the system by the blood, and general symptoms ensue. These include rigors, considerable fever, and perhaps vomiting. Death occurs in about a week, from the constitutional condition, or it may be due to œdema of the larynx. Recovery, however, may take place.

Internal anthrax includes a bronchial and a gastro-intestinal variety. The former is *wool-sorter's disease*, which is caused by the inhalation of infected hair or dust from the skins of animals. There is fever at the outset, and the pulse is rapid and feeble. Moist and dry râles, and a blood-stained sputum, are the principal physical signs, and are in no way proportional to the intense prostration. Death may take place in a few hours or after several days ; but if the patient survives for a week, he will probably recover. After death the pleural and pericardial cavities are found to contain clear, non-inflammatory fluid ; the lungs are collapsed ; the respiratory mucous membrane is swollen ; the bronchial glands are enlarged ; and hæmorrhages are present in various parts. There is often no splenic enlargement.

The *gastro-intestinal* variety of the disease results from infection by the alimentary tract, and is characterised by abdominal pain, vomiting, and diarrhœa, in addition to the general symptoms. The spleen is enlarged.

Diagnosis.—The local lesion, as described above, is characteristic, and it is single. The patient's occupation may suggest the diagnosis. Serum from a vesicle should be examined by staining for the bacillus. If there is no recognisable local lesion, a mouse may be inoculated with blood from the patient ; if the disease is anthrax, the animal will die in a few days, and the bacilli will be found in its blood.

Prognosis.—According to Newton Pitt, the mortality at Guy's Hospital, which receives most of the London cases, is about 20 per cent. when the pustule is on the head or neck, and 10 per cent. when it is on the hand. Symptoms of general infection, and in particular a rapid, feeble pulse, are ominous ; but even patients whose urine contains bacilli may recover.

One attack does not confer complete immunity against another.

Treatment.—Until recently the best practice was to excise the pustule, and then to cauterise the raw surface with strong carbolic acid, or to pack the wound with iodoform, or to apply powdered ipecacuanha. (Ipecacuanha powder

has the property of destroying anthrax bacilli, and it has been largely used internally as well as locally in this disease.) Carbolic lotion was injected several times a day into the surrounding tissues. To judge from published records, however, much better results can now be obtained by the use of Sclavo's anti-anthrax serum, of which 30 or 40 c.c. should be injected subcutaneously. The dose should be divided between three or four different parts of the abdominal wall, and may be repeated on the following day if need be. In grave cases 10 c.c. may be injected into a vein on the back of the hand. The serum treatment appears to render excision of the pustule unnecessary. The œdema may increase after the injection, though the patient's condition is improving in every other way. This is perhaps due to the liberation of the intracellular toxin from the bodies of the dying bacilli in the local lesion. Discharges and dressings from the case must, of course, be thoroughly disinfected.

36. GLANDERS

(Farcy. Malleus. Equinia).

Definition.—An infectious disease of the horse and ass, due to a specific micro-organism, sometimes communicated to man, and characterised by the development and breaking down of multiple granulation-tissue nodules in the nose (glanders) or under the skin (farcy).

Etiology.—The *Bacillus mallei* resembles the tubercle bacillus in appearance, but is rather shorter and thicker.

In mankind the disease occurs chiefly in grooms, knackers, and others who work with horses, and is usually directly inoculated into some breach of continuity of the skin or mucous membrane. But infection can take place through sound skin or mucous membrane. Fatal accidental inoculations have sometimes occurred in the laboratory, and the disease has occasionally been transmitted from man to man.

Morbid Anatomy.—Two forms of the disease are recognised, but these are frequently associated in one individual.

The elementary lesion in either case is a granuloma which contains the specific bacilli in and between the cells of which it consists. In *glanders* the granulomata develop in the nasal and respiratory mucous membrane, where they break down and give rise to ulcers. In *farcy*, the granulation-tissue nodules ('farcy buds' or 'buttons') are in or under the skin—viz., in the lymphatic vessels and glands—and there they tend to suppurate. Whichever type appears first, the other usually follows, so that the two are often met with in the same case.

After death the blood is found to be fluid. The muscles are soft, and contain hæmorrhagic abscesses. Abscesses are also found in the parotid, cervical, and other glands. Nodules may be found in the liver, spleen, and other internal parts, and pneumonia may be present in patches.

Anatomically, therefore, the disease bears a resemblance to pyæmia.

Incubation.—The incubation period is usually from one to eight days, but may extend over weeks.

Symptoms.—Both glanders and farcy may be acute or chronic. In *acute glanders*, the invasion is characterised by shivering, headache, prostration, and fever. Pains in the joints and muscles are troublesome, and are suggestive of rheumatism. The nose becomes swollen, red, and painful, and discharges a dirty, semi-purulent, and often blood-stained material. An eruption appears on the face and trunk, and may suggest small-pox. It begins in the form of minute red spots which resemble flea-bites. These develop into shotty papules, each of which lies in a little depression in the corium. The papules give place to vesicles, and these in turn to pustules, which, if close to one another, become confluent. The surrounding skin has an erysipelatous aspect. The pustules may give rise to ulcers, and if similar lesions develop in the deeper tissues, serious sloughing may result.

Pneumonia, suppurative arthritis, etc., may also occur. The duration is from one to two weeks, and the issue is almost always fatal.

In *chronic glanders*, the symptoms are less severe, the

lesions fewer, and the course protracted over weeks or months. The condition is apt to be mistaken for chronic coryza. Recovery occasionally takes place.

In *acute farcy*, the nose escapes, and the eruption suggestive of small-pox is uncommon. The disease resembles pyæmia. It is usually due to inoculation of the virus into the skin, and the result is severe inflammation at the place of infection, with involvement of the lymphatics, and multiple abscesses in the subcutaneous and muscular tissues. Arthritis also occurs. The patient generally dies in a week or two.

Chronic farcy may go on during months or years. The nodules break down into abscesses. The disease may end in recovery, or may give place to acute glanders.

Diagnosis.—The patient's occupation, and any known exposure of a wound to the risk of infection, are important. In chronic cases the diagnosis may be very difficult. Mallein (prepared by filtration of a glycerin veal-broth culture of the bacillus) is injected for diagnostic purposes into suspected animals; if the animal is diseased, a reaction ensues. In the case of man also, mallein may be used, or the discharge from a lesion may be introduced into the abdominal cavity of a male guinea-pig. If glanders is present, the testes of the guinea-pig will in the course of a few days be found to be the seat of characteristic changes. It is very difficult to find the bacillus by microscopic examination of the discharges.

Prognosis.—This is always grave. Almost all acute cases die. It has been said that about half of the chronic cases recover. The more chronic the course, the better the outlook.

Treatment.—The general health must be kept up as much as possible. Accessible lesions should be dealt with by modern surgical methods, and discharges should be disinfected. Repeated inoculations of mallein frequently lead to recovery in horses.

37. HYDROPHOBIA (Rabies. Lyssa).

Definition.—An acute specific infection, involving specially the central nervous system, and usually communicated to man by the bite of an affected carnivorous animal.

Etiology.—The specific organism has not yet been identified, though Negri has described as present in the central nervous system bodies which may be protozoa. The virus is present in the central and peripheral nervous system, and in the saliva and other secretions, but not in the blood or urine.

The disease may be communicated to many animals besides the carnivora. Proper muzzling of dogs, in countries where there are no wolves, would almost certainly eradicate the disease. Thus in recent years the muzzling order has completely stamped out the disease among human beings in England, whereas during the preceding half-century no year was without its mortality. Bites on exposed parts are more dangerous than bites on covered parts, as the clothing often wipes the virus off the teeth. It is stated by Horsley that of those bitten by rabid dogs, not more than 15 per cent. suffer. In the case of wolf-bites, the percentage is several times as h gh. Cats are more dangerous than dogs, but not so dangerous as wolves.

Morbid Anatomy.—There is usually congestion about the throat. In most cases there are microscopic changes in the central nervous system, and especially in the medulla oblongata. They include dilatation of small vessels, accumulations of round cells around the vessels and nerve-cells (especially the motor ganglion cells), minute hæmorrhages, and degenerative changes in the nerve-cells. The leucocyte accumulations have been termed "rabic tubercles." Collections of lymphoid and endothelioid cells have also been found round the nerve-cells of the sympathetic and other ganglia.

Incubation.—The incubation period varies greatly (two weeks to one and a half years), but in man the average may be stated as six weeks. The wound usually heals in the ordinary way.

Symptoms.—Towards the end of the incubation period, there may be some discomfort in the region of the bite, and at the invasion this may be replaced by considerable pain, but local symptoms are often absent. Among the first symptoms (premonitory stage) are malaise, mental depression, and a dread of impending danger. Then there ensues great difficulty in swallowing and breathing. An attempt to swallow causes spasm of the pharynx. Indeed, any afferent stimulus causes a very painful reflex spasm, which specially involves the muscles about the mouth and throat. This increases, and after a time spreads to the respiratory muscles, including those concerned in forced inspiration. As the severity of the spasm increases, so does the readiness with which it is induced, and a slight draught of air may excite it. The difficulty in swallowing the saliva may lead to frothing at the mouth, and also to the peculiar cough supposed to resemble the bark of a dog. The spasm continues to spread over the body and becomes convulsive in character. The temperature is elevated. General hyperæsthesia is present, and delirium or actual mania may set in. Death may now occur from asphyxia or syncope. In other cases the convulsive attacks cease, and a state of exhaustion is present for some hours before death. The different stages recognised by some (depression, excitement, paralysis) are often not well defined. The usual duration is from two to four days.

In the lower animals, two types of the disease are recognisable. The more common is ' furious rabies,' characterised by great agitation with hallucinations, and later on by paralysis. The other type, ' dumb rabies,' seen specially in rodents, is characterised by paralysis without any preceding stage of excitement.

Diagnosis.—The suspected animal ought to be kept in safe isolation to see whether it is diseased ; and if, after a time, there is still doubt, its medulla may be inoculated into a rabbit. The incubation period is longer than that of tetanus, and there is no early trismus. Respiratory spasm, difficulty in swallowing, and extreme dread, are prominent features of hydrophobia.

Lyssophobia, or *pseudo-hydrophobia*, is a group of hysterical symptoms seen in persons who have been bitten by a dog, whether mad or not, and who are in terror lest they take hydrophobia. The most important diagnostic points are the absence of true respiratory spasm, and the improvement which takes place if the sufferer's anxiety can be relieved. The temperature is normal, and the duration may be considerably longer than that of genuine hydrophobia.

Prognosis.—After the disease has definitely set in, the case is hopeless.

Treatment.—The bite should be promptly cleansed and cauterised. Pasteur's method of preventive inoculation should be utilised as early as possible, the patient receiving successive subcutaneous injections of the virus in increasing intensity. The injections are emulsions made from spinal cords of rabbits which have had the disease. Drying the cords in air for varying periods diminishes their virulence proportionately. By this treatment the mortality of bitten persons has been reduced almost to the vanishing point, and the procedure is quite free from danger. It occupies a fortnight, and it ought to be begun within a week after the bite.

If the disease has developed, the patient should be kept in a darkened room, and an attempt should be made to relieve the spasms by pushing such remedies as chloroform, morphine with chloral, and curare.

38. FOOT-AND-MOUTH DISEASE

(Aphthous Fever. Aphthæ Epizooticæ. Epidemic Stomatitis).

Definition.—An acute infectious fever associated with an eruption of vesicles on the mucous membrane of the mouth, and sometimes on the skin of the hands, feet, and other parts.

Etiology.—The virus has not yet been identified with certainty, but Siegel has described a parasite which closely resembles that of small-pox, and is likewise regarded as a

sporozoon.[1] The disease is chiefly one of cattle, sheep, and pigs, among which it spreads with great rapidity; but it may be transmitted to man, either by inoculation of a surface lesion, or by the use of contaminated milk, butter or cheese.

Incubation.—The incubation period is probably a week or less.

Symptoms.—Among the early symptoms are shivering, fever, and a sense of heat in the mouth and extremities. After a few days the eruption appears in the mouth, and perhaps also on the hands and feet. The vesicles in the mouth are usually scanty and soon burst, leaving ulcers which heal in the course of a week or two. In severe cases there may be great disturbance of the digestive system, but a fatal termination is quite exceptional.

Diagnosis.—The distribution of the eruption is usually sufficient for the distinction from chicken-pox.

Treatment.—Borax and glycerin may be applied to the mouth at short intervals. Painful or obstinate ulcers should be touched with powdered alum or solid nitrate of silver. When an epidemic is prevailing among cattle in the district, the milk should be boiled.

39. DYSENTERY.

Definition.—A designation applied to certain kinds of intestinal flux, of which the acute forms are characterised by inflammation of the large bowel and by frequent evacuations containing blood and mucus, while the chronic forms are characterised by ulceration of the bowel and by alternations of diarrhœa and constipation.

Etiology.—Two at least of the varieties of dysentery are attributable to specific causes. Apart from these, there are other factors in the etiology of the disease. It is endemic in many parts of the tropics, and occurs in epidemic form in temperate climates. Thorough-going sanitation tends to abolish it. It is often a plague to armies on campaign, and here a low temperature at night, unsuitable food, and

[1] *British Medical Journal*, October 21, 1905, Epit., p. 64.

bad drinking-water may each take a part in the causation. It may break out in overcrowded asylums. Malaria is often associated with dysentery, and is perhaps one of the causes. Alcoholism is a predisposing cause.

Amœbic dysentery is due to the *Amœba dysenteriæ*, which was first described by Lambl in 1859. This protozoon belongs to the class *Rhizopoda*, and has a diameter of from 15 to 20 μ, or two or three times that of a red blood corpuscle. It possesses a clear ectosarc, a granular endosarc, a nucleus, and one or two vacuoles. It shows movements like those of the common amœba, and it frequently contains red blood corpuscles. It is found not only in the stools, but also in the tissues at the seat of lesion in the bowel, and in the pus and walls of the frequently associated hepatic abscess. It can be cultivated from the stools and intestinal ulcers, and it can be recognised in the tissues by staining. Amœbic dysentery may be either acute or chronic. It prevails extensively in the tropics, and occurs in sporadic form in temperate climates. Infection takes place through drinking water or eating vegetables or other articles which have been contaminated.

Bacillary dysentery (*acute specific dysentery*) is due to the *Bacillus dysenteriæ*, discovered by Shiga in 1898 in connection with a serious form of the disease which has long prevailed in Japan, but apparently described by Chantemesse and Widal in 1888. The severe epidemics of acute dysentery which occur in tropical regions are generally due to this organism, but it is also met with in temperate climates. It has, moreover, been found in the summer diarrhœa of infants. Different races of the bacillus are known to exist, characterised by slight differences in their agglutination with immune serum, and in their action upon different sugars. The bacillus is found in the stools, but the mode of infection has not yet been ascertained. It agglutinates with the blood-serum of patients, and with the serum of immunised animals.

A third form of dysentery, which is not so obviously specific, is the *acute catarrhal* dysentery or *acute ileo-colitis* of temperate regions. Possibly, however, this may prove

to be a sporadic form of the bacillary disease. A fourth form is *diphtheritic* dysentery, which may be either primary, or secondary to such affections as Bright's disease and pneumonia. The specific bacillus has been found in association with this variety.

To search for the *Amœba dysenteriæ* the stools should be examined soon after evacuation. One of the little masses of mucus should be smeared on a cover-slip, and examined on a warm stage at a temperature of about 100° F. Changes of shape and of position constitute a striking characteristic of this as of other amœbæ. In a cool atmosphere it becomes non-motile. The *Bacillus dysenteriæ* should also be looked for in the particles of mucus.

Morbid Anatomy.—The lesions are mainly in the large intestine, though the lower part of the ileum is sometimes involved. In the *amœbic* form elevations appear above the general level of the mucous membrane. These are due to œdema and to proliferation of the tissue-cells. Then the overlying mucosa dies, and the infiltrated submucosa is exposed as a grayish-yellow mass, which subsequently sloughs away. The resulting ulcer often has its edge undermined, and it may penetrate as far as the serous coat of the bowel. The microscope shows amœbæ and proliferation of the tissue-cells, but remarkably few polymorphonuclear leucocytes. As the ulcers heal, strictures may be produced. The liver may contain scattered foci of tissue necrosis, and in a number of cases abscesses, either single and large, or multiple and small. In *bacillary* dysentery, the mucosa of the large bowel is swollen, red, and thrown into coarse elevations. It may be the seat of hæmorrhage, and there is often a thin layer of necrosis on its surface, but there is no ulceration. The solitary follicles are red and swollen. The ileum may share in these changes. In severe cases the mucosa may be greatly thickened, and in some parts gangrenous.

In *acute catarrhal* cases the sigmoid flexure, rectum, and descending colon suffer most. The mucous membrane is congested and swollen, and covered with tough, bloody mucus. The solitary follicles are acutely inflamed, and may

suppurate. In *diphtheritic* cases there is an exudation on the top of the folds of the large intestine.

In *chronic* dysentery various changes may be met with in the bowel, including chronic ulceration, pigmentation, and cicatrisation of the mucous membrane, hypertrophy and atrophy of the wall, and dilatation and narrowing of the lumen.

Incubation.—The period of incubation in bacillary dysentery is said to be not more than forty-eight hours.

Symptoms.—*Acute* dysentery may set in suddenly, or in the course of what was regarded as simple diarrhœa. There are frequent stools, which contain much mucus and some blood. Griping pains in the abdomen (tormina) and a constant desire to go to stool (tenesmus) are present. The tongue becomes furred. Considerable fever may be present at the outset of the bacillary type, but in amœbic dysentery it is usually slight, and may be absent. There may be tenderness over the sigmoid flexure and a burning sensation at the anus. After a few days or weeks the attack subsides, or it may pass on to ulceration, or again it may become subacute or chronic.

If ulceration takes place, the stools are not only mucous and bloody, but also contain little shreds of slough, and have a foul odour. This form is more protracted than the simple variety, but may subside in time, though it, too, may become chronic. In the gangrenous variety large sloughs may appear in the stools, and these smell horribly. The abdomen is distended and tender, fever is considerable, and the typhoid state may supervene and lead to a speedy death. Or death may be due to hæmorrhage, peritonitis, or syncope. The most virulent forms of dysentery, whether amœbic or bacillary, may cause death in from four to seven days.

Chronic dysentery is a frequent result of an acute attack. While bacillary dysentery sometimes assumes a *subacute* phase which may continue for many months, amœbic dysentery, whether acute or subacute to begin with, is frequently continued as a *chronic* illness. The stools regain somewhat of the fæcal character, but still contain

mucus and sometimes blood. There may be tenderness over the sigmoid flexure or other part of the large bowel. Constipation may alternate with diarrhœa, and exacerbations may result from exposure or indiscretions in diet. After months or years this may tell seriously on the general health ; the tongue becomes red and raw, and death often results directly or indirectly from the cachectic condition.

Complications and Sequels.—The most important is hepatic abscess. Occasionally there is local peritonitis. Perforation occasionally takes place, and leads to general peritonitis, perityphlitis, or periproctitis. Malaria may be associated with dysentery. Other complications or sequels are arthritis, inflammations of serous membranes, pyæmia, chronic Bright's disease, and paralysis.

Diagnosis.—The acute form is generally easy of recognition. In the diphtheritic and gangrenous forms, the early appearance of blood in the stools, and the absence of spots and of Widal's reaction, may assist in excluding enteric fever. In doubtful cases the rectum should be examined. In the amœbic form the amœba is found in the stools. The other specific form is recognised by the marked fever, by the bacillus in the stools, and by that bacillus agglutinating with the patient's blood-serum (though this reaction may not appear for a week or two).

Prognosis.—Malignant or gangrenous dysentery is almost invariably fatal. There is a considerable mortality from the other acute types, but in the tropics the disease is not nearly so fatal when it attacks Europeans as when it attacks natives. Much depends upon the nursing and treatment obtainable. Many acute cases become chronic.

Treatment.—Rest in bed is important. The diet must be light, and given in small quantities at a time. Milk, whey, barley-water, rice-water, chicken-tea, and weak tea may be given. At the commencement, particularly if no diarrhœa has preceded the attack, it is well to clear out the bowels by a purge, either of salts or of castor oil with opium. In addition to these measures, ipecacuanha may be tried. The patient gets no food or drink for three hours before the dose. Twenty minutes before the dose he gets morphine

hypodermically, or laudanum by the mouth, and about the same time a mustard-plaster or poultice is applied to the epigastrium. The dose consists of 30 grains of freshly-powdered ipecacuanha root. The poultice is removed a few minutes later, but the patient continues to lie flat on his back for several hours afterwards. If the powder is vomited, the dose should be repeated after some hours, and, in any case, smaller doses should be continued for several days. Another method of treatment is by sulphate of sodium or of magnesium, given in drachm doses every quarter of an hour till the stools become feculent. Another mode of treatment is by mercury—*e.g.*, $\frac{1}{100}$ of a grain of the perchloride every two hours, or 5 grains of calomel three or four times a day. Bismuth in large doses (1 to 2 ounces daily) is sometimes of service. Morphine may be required to relieve pain and restlessness, and benefit may be obtained from the firm application of a cold wet pack to the abdomen. After the stools become feculent, astringents, such as lead and opium, may be exhibited. Irrigation of the large bowel is theoretically good treatment, but is scarcely to be recommended in acute cases on account of the pain and the great irritability of the bowel. It may, however, be tried in chronic cases, though here, too, trouble may be experienced. The enema should amount to several pints, and may contain silver nitrate (20 or 30 grains to the pint), lead acetate, or some other astringent. Osler speaks highly of quinine used in this way in amœbic dysentery ; the strength is 1 in 5,000 to begin with, and may be increased. In chronic cases, rest in bed should be tried for a time ; later on, a sea-voyage may be beneficial. Castor oil in small doses, and bismuth in large doses, in addition to irrigation of the intestine, deserve a fair trial.

Serum from immunised horses has been employed in the treatment of the endemic dysentery of Japan, and also in the treatment of bacillary diarrhœa in infants. In neither case, however, do the results appear to be very encouraging.

40. MALARIAL FEVER[1]

(PALUDISM.[2] MARSH, JUNGLE, ROMAN, OR AFRICAN FEVER).

Definition.—A group of specific infections, characterised by continuous, intermittent, or remittent pyrexia, splenic enlargement, anæmia, and pigmentary deposits.

Etiology.—Malaria occurs in parts of Italy and the United States, and prevails in the tropics. It is specially serious in tropical Africa. It is now almost extinct in Britain, but before the days of efficient drainage it was an important disease.[3] A marshy soil and a high mean temperature favour it by favouring the mosquito, and possibly also the specific parasite. In the tropics, the disease prevails mostly in spring and autumn ; in temperate regions like the United States, chiefly in autumn, and very slightly in spring.

The parasite, which was discovered by Laveran in 1880, belongs to the class sporozoa of the protozoa or lowest animal forms, and is thus nearly related to the coccidia. It is known as the *Plasmodium malariæ*, the *Hæmamœba malariæ*, or the hæmatozoon of malarial fever. For a complete cycle of existence the parasite requires two hosts ; and there are three species of parasite which find their intermediate host in man, and their definitive host in a mosquito belonging to the genus *Anopheles*. These three species of parasite are associated respectively with tertian, quartan, and æstivo-autumnal fever. Patients often have two kinds in their blood at one time. Each individual parasite enters into, lives and develops in, and eventually destroys, one red corpuscle.

[1] Ital., *mala aria* = bad air. [2] Lat., *palus* = a marsh.

[3] Sir Thomas Browne, writing from Norwich to his son Edward, about 1679, says : 'There have been, and are still, many quartane agues.' He narrates the case of a lady who in her pregnancy suffered from quartan fever, and whose new-born child was seriously affected by the same disease ('Works,' Wilkin's edition, i., pp. 227, 228).

James I. of England and Oliver Cromwell are said to have died of malarial fever.

In *tertian fever* the parasite (*Plasmodium vivax*) first appears in the red corpuscle as a small colourless body of varying shape. After some hours it is seen to be larger and to contain granular pigment. Moreover, it shows active amœboid movements. After twenty-four hours it almost fills the swollen corpuscle, and contains much pigment. This is the full-grown parasite, which may become either (*a*) a sporocyte or (*b*) a gametocyte.

(*a*) In the case of a sporocyte, segmentation takes place, and the parasite is divided up by lines which pass from circumference to centre, into from fifteen to thirty different segments, which are young spores. Finally, the red corpuscle gives way, and spores and pigment are set free in the blood, with the result that a febrile paroxysm ensues. The spores attack fresh red corpuscles, and the asexual or fever-producing cycle just described is repeated.

(*b*) The gametocytes do not undergo segmentation, but constitute sexually differentiated bodies, which require the mosquito for further development,—the sexual or mosquito cycle. When the mosquito bites an individual whose blood contains these sexual forms, fecundation of the female element takes place in the insect's stomach. After reaching the insect's stomach, the male and female gametocytes escape from the red corpuscles. The male now gives off spermatozoa (microgametes, flagella) which detach themselves, and enter into and fertilise the female gametocyte, which now becomes an ookinet or zygote. The zygote penetrates to the outer muscular layers of the stomach wall, grows rapidly, and is converted into a kind of capsule, which contains vast numbers of slender, spindle-shaped bodies called ' blasts ' or ' sporozoites.' The capsule now ruptures, and sets free the young sporozoites into the body cavity of the insect. The sporozoites find their way through the thorax into the cells and ducts of the salivary glands, whence they are injected into the blood of any human being whom the insect bites.

The cycle in the blood, ending with the liberation of spores and pigment, takes forty-eight hours, and its completion corresponds to the febrile paroxysm. If all the

parasites mature simultaneously, the fever is tertian. If there are two sets, maturing on alternate days, it is double tertian (one form of quotidian fever).

The parasite of *quartan fever* (*Plasmodium malariæ*) is on the whole similar, both in sexual and asexual phases, to that of tertian fever, but its outline is more definite, its movement less, its pigment-granules coarser and darker, its segments only six to twelve in number, and its cycle of seventy-two hours' duration. If the parasites mature simultaneously, the fever is quartan ; if one set matures a day later than the rest, it is double quartan ; and if there are three sets, maturing at intervals of a day, it is triple quartan (a second form of quotidian fever).

The parasite of *æstivo-autumnal* or *malignant tertian fever* (*Plasmodium præcox*) is much smaller and much less pigmented than the others, and the containing corpuscle becomes crenated and wrinkled. The cycle extends over about forty-eight hours, but may vary to some extent. Several groups of parasites may be present, so that the fever may be irregular. Only the earliest stages of the sporocyte or asexual phase are observable in the peripheral circulation, since the later stages (segmentation, etc.) are passed through in the blood of internal organs, and especially the spleen and bone-marrow. After about a week, crescent-shaped bodies, containing central clumps of pigment, appear in the peripheral blood. These are sexual forms (gametocytes), and cannot sporulate in the human host ; but on reaching the stomach of the mosquito the male elements give off long motile flagella, which become detached, and penetrate and fecundate the female elements, after which the cycle is continued as already described.

That the mosquito might be the carrier of the disease was suggested by Manson and others some years after Laveran's discovery of the parasite in the blood, and was demonstrated as a fact by Ronald Ross in 1895-1899. Men have been infected experimentally. Thus Manson in 1900 infected two men in London by mosquitoes brought from Italy. On the other hand, by living in mosquito-proof houses men will escape infection in the most notorious haunts of malaria.

So far as our present knowledge goes, it appears that the mosquitoes in which the parasite of human malaria develops are confined to the genus *Anopheles;* that all species of that genus may act as hosts; and that the parasite in question exists only in man and in *Anopheles*. In the tropics most of the natives and many old residents have the parasites constantly present in their blood, and if their health should be slightly reduced in any way they are liable to a relapse of malarial fever. Native children especially seem to become readily accustomed to the presence of the parasite and to remain free from symptoms. At the same time, they are a very important source of infection for mosquitoes, and through these for other human beings. Travellers therefore run great risk by camping near or living in native villages.

The female *Anopheles*, two or three days after her meal of blood, lays her eggs on water, chiefly in stagnant puddles on the ground, or in sluggish streams, and usually quite near the house where she obtained the human blood. After depositing the eggs, she returns to repeat the process of sucking blood, sleeping, and laying eggs. She bites chiefly at night, and is comparatively noiseless, so that her presence may quite escape notice. The one reason why malarial fever prevails near stagnant water is that *Anopheles* breeds in such water.

Both sexes of the mosquito live on fruit, but it would appear that only the female sucks blood. Ross thinks that she requires a meal of blood before she can lay her eggs. *Anopheles* conveys elephantiasis as well as malaria.

Morbid Anatomy.—In acute attacks the spleen is enlarged and soft (' acute splenic tumour ') ; the swelling occasionally sets in so rapidly as to rupture the capsule. In chronic cases the spleen is large and firm (' chronic splenic tumour,' ' ague-cake '), and of a dark gray colour from pigmentation of the trabeculæ, vessel walls, and phagocytes. The liver and kidneys may be enlarged, and these organs, as well as the brain, bone-marrow, and mucous membrane of the alimentary tract, are dark in colour through pigmentation. The pigment is melanin, a substance which, though derived

from hæmoglobin and containing iron, does not give the ferrocyanide reaction.

Incubation.—The incubation period in the mosquito is from one to two weeks after the bite ; in man it varies, according to Ross, from about six to twenty days.

Symptoms.—1. *Intermittent Fever* or *Ague.*—This includes quotidian, tertian, and quartan, the first being either double tertian or triple quartan. The paroxysm of ague consists of three stages—the cold, hot, and sweating.

At the commencement of the *cold* stage there is lassitude, with headache, yawning and stretching, and sometimes sickness. Then a violent rigor sets in, and though the skin looks and feels cold, the axillary temperature may rise to 105°, or higher. This stage lasts from a few minutes to an hour.

In the *hot* stage the skin is dry, and looks and feels hot. Thirst, headache, and sometimes delirium are present. This stage lasts for an hour or two.

The *sweating* stage also lasts for a couple of hours, and is characterised by free perspiration, with relief from all symptoms.

In the paroxysm the spleen is enlarged. In the interval the patient feels well. If the disease is not treated, the continued recurrence of paroxysms leads to anæmia, and in chronic cases the malarial cachexia develops.

2. *Remittent, Irregular, Continued,* and *Æstivo-autumnal Fevers* (due to the malignant or æstivo-autumnal parasite).—There may be malaise at the outset for some days, or there may be a rigor at once. The fever continues for some time —frequently for one or for two weeks. The temperature remits, and with the remissions there may be slight sweating ; but there is no intermission. The general appearance of the patient, as well as the continued or remittent fever, the splenic enlargement, and the occurrence in autumn, may be very suggestive of enteric. There may be jaundice and slight delirium. If vomiting is severe, the fever is sometimes styled *bilious remittent.*

Irregular types of fever connect the remittent with the intermittent types. As compared with intermittent fever

(ague), the paroxysm is longer, the rise and fall of the temperature are often gradual, and the periodicity is less regular.

3. *Pernicious Malarial Fever* (due to the æstivo-autumnal parasite) is characterised by symptoms pointing to serious involvement of some organ or system. Thus, in (*a*) the *comatose* form, there is high fever, with coma or delirium, and the patient may die in the first or in a later attack. In such cases, thrombi of parasites have been found in the brain, with serious changes in the surrounding tissues. In (*b*) the *algid* form, there are vomiting, diarrhœa, and intense prostration, almost as in cholera. Here the alimentary mucosa may be the seat of special invasion by the parasites, with consequent thrombosis and ulceration. (*c*) *Blackwater fever* is believed by some to be malarial (*malarial hæmoglobinuria*); but others hold that it is a distinct specific infection; while a third view is that it results from the administration of quinine. The evidence is against the idea of a separate infection distinct from that of malaria, and in favour of quinine being in some mysterious way a cause of hæmoglobinuria in malarial subjects. Koch thinks that it may be induced by the sudden commencement of very large doses of quinine in malarial subjects who have long neglected the drug altogether.

Black-water Fever (*Malarial Hæmoglobinuria, Hæmoglobinuric Fever* or *West African Fever*) is one of the commonest causes of death among Europeans in tropical Africa, and is a terrible scourge on great parts of the West Coast. It occurs only in malarial subjects, and the malarial parasite is usually, though not always, to be found in the blood. It seldom occurs till the individual has been for at least a year in the endemic area and has suffered much from malaria, and it is almost confined to those who have neglected continuous treatment. One attack strongly predisposes to another. Hæmoglobinuria, with the usual constitutional symptoms, occurs in the course of what the patient probably regards as one of his ordinary febrile relapses. High fever and vomiting are prominent symptoms, and the urine is sometimes almost black. Jaundice usually supervenes.

Death results in about a fourth of the attacks. Of persons attacked, more than half may succumb, but many individuals live through several attacks.

The *malarial cachexia* is characterised by splenic enlargement ('ague-cake'), and by anæmia with the usual symptoms, viz., pallor, shortness of breath, œdema, etc. With proper treatment most cases ultimately recover.

Sequels.—Malaria is sometimes a factor in the causation of leucocythæmia and of Raynaud's disease. Certain forms of paralysis have been referred to it. Orchitis has also been observed.

Diagnosis.—In many cases this is simple. In the æstivo-autumnal forms, which resemble enteric, Widal's test should be employed, and the blood should be examined microscopically with a $\frac{1}{12}$-inch oil immersion lens. Staining is not necessary for the detection of the parasite, but, if it is desired, Manson's procedure may be followed. The blood is received at one end of a piece of guttapercha tissue or tissue-paper, 2 inches by 1 inch. The tissue is then laid on a cover-slip, and after a few seconds drawn horizontally along it, leaving an excellent film. This is allowed to dry, and is then fixed by absolute alcohol, which should remain on it for five minutes. It is again dried, and then stained for thirty or forty seconds with methylene blue (2 per cent.) and borax (5 per cent.) in distilled water. The film is then washed, dried, and mounted in xylol balsam.

Leucocytosis is absent in malaria, and, as Osler remarks, an intermittent fever which resists quinine is not malarial.

Prognosis.—In the benign fevers the chief risk is that repeated attacks will induce cachexia. Repeated attacks of remittent fever may lead to death, and pernicious fever is, of course, very dangerous. Hæmoglobinuric fever is also dangerous.

Treatment.—To avoid infection in a malarial district, care should be taken to avoid mosquito-bites, and quinine may be taken in daily doses of 5 or 10 grains. The many scientific men who have been engaged in recent years in the study of malaria have generally escaped serious illness, and in most cases infection, because they have taken

measures to prevent mosquitoes from biting them. The adult insects can be destroyed by a blow ; the larvæ can be destroyed by emptying the puddles in which they live, or by spreading a film of thin oil on the surface of the water, so as to prevent the creatures from breathing. The breeding-places should be removed, if possible. But it is the duty of every person living in an infected region to have himself thoroughly protected at night by a mosquito-net, and in specially dangerous places it is well, in addition, to live in a mosquito-proof house.

During the paroxysm the patient should rest in bed, and in the cold stage may swallow hot drinks. Quinine is the specific remedy ; it destroys the parasites most effectually when they are free in the blood and not inside the red corpuscles. Before it is taken, the liver should be unloaded by a mercurial purge.[1] 20 to 30 grains of quinine may be taken in solution daily for a fortnight, 15 grains daily for the second fortnight, 10 grains daily for the second month, and smaller doses for two months more. If a relapse occurs, treatment should be begun again from the beginning. In æstivo-autumnal fevers larger doses may be required, and in pernicious attacks ½-drachm doses (dissolved with 5 grains of tartaric acid) should be given hypodermically every few hours, to produce results as quickly as possible. Stimulants may be required, and opium also may be useful. Patients with cachexia should resort to a non-malarial district at a high altitude, and should take arsenic internally. In cases of hæmoglobinuric fever quinine should be avoided in the paroxysm, but should be given in the interval if the malarial parasite is found in the blood.

[1] The following was Dr. Livingstone's prescription : Resinæ jalapæ, pulv. rhei, āā gr. vi.-viii. ; calomel, quinin., āā gr. iv. Make into pills with spirit of cardamom, and after they have taken effect, give 4 grains of quinine every two hours till deafness or ringing in the ears sets in.

41. TROPICAL SPLENOMEGALY

(Tropical Cachexia. Dum-Dum Fever. Kala-azar).

Definition.—A chronic infection due to a protozoon, and characterised by splenic enlargement, anæmia, irregular fever, hæmorrhages, and emaciation.

Etiology.—The parasite of this disease was discovered by Leishman in 1900 in the spleen of a soldier invalided home from Dum-Dum for tropical cachexia with enlarged spleen. The disease itself had been known in India for many years as a cachexia which did not yield to quinine. It was called 'Dum-Dum fever' by some observers, and 'non-malarial remittent fever' by Crombie. The parasite, known as the 'Leishman-Donovan body,' is found in abundance in the spleen, but it has also been found in the liver, bone-marrow, and mesenteric glands, and occasionally in the peripheral blood.[1] In a smear taken from the spleen and stained, it is seen to be round or ovoid in form, 2 or 3 μ in diameter, bounded by a cuticle, within which there are enclosed two masses of chromatin, the one large and circular, the other small and rod-shaped. Different views have been expressed as to the nature of this protozoon, Leishman regarding it as a trypanosome,[2] Laveran thinking it a species of piroplasma, and Ross suggesting that it is a tissue parasite belonging to a hitherto unknown genus. The parasites can be obtained during life by drawing blood from the spleen. Many of them are contained in large cells which are apparently endothelial and phagocytic in their nature.

The disease prevails widely in India and other tropical countries.

Morbid Anatomy and Symptoms.—The onset may be abrupt or insidious. It begins with fever, or with acute

[1] The same parasite has been found in the granulation tissue of tropical ulcer, Delhi boil, Aleppo button, Oriental sore, etc.

[2] Rogers claims to have developed trypanosomes in cultures of Leishman-Donovan bodies from the splenic blood of a case of kala-azar; but Christophers, while admitting that these are flagellates, denies that they are trypanosomes.

digestive symptoms, or with pneumonia, or with gradually increasing debility accompanied by pain in the limbs and splenic enlargement. The spleen is always, and the liver frequently, enlarged. The skin becomes earthy-looking, and emaciation takes place. Irregularly remittent fever may persist for many months. There may be hæmorrhage from the mucous membranes. A secondary anæmia develops, with leucopenia and a relative increase in the non-granular leucocytes.

The disease is met with at all ages and in both sexes.

Diagnosis.—This depends on the examination of blood drawn from the spleen during life.

Prognosis.—The duration varies from a few months to several years. At the outset of an epidemic the case-mortality may be as high as 90 per cent., but in the course of a few years the virulence of the disease abates very greatly. The ordinary mortality has been put at from 25 to 35 per cent.

Treatment.—Quinine is not a specific in this disease, but should, nevertheless, be administered along with other tonics.

42. TRYPANOSOMIASIS (Sleeping Sickness).

The trypanosomes[1] belong to the *mastigophora*, a flagellated class of the *protozoa*. They possess a longitudinal, wavy membrane, whose thickened border ends anteriorly in a flagellum; and they divide longitudinally. They are about 20 μ long, and in fresh blood are actively motile. The first trypanosome to be discovered in a mammal was one which was found by Lewis in the rat (*T. Lewisi*) in 1877. This parasite is supposed to be transmitted by a flea, and is not pathogenic. In 1880, Evans discovered in the blood of horses, mules, and camels affected with *surra* in India, the trypanosome (*T. Evansi*) which causes that disease. In 1896, Bruce showed that the deadly *nagana* or *tsetse-fly disease* of horses in South-East Africa was due to a trypanosome (subsequently named *T. Brucei*),

[1] Screw-like bodies: Greek, τρύπανον, borer; σῶμα, body.

and that it was transmitted by a biting fly (*Glossina morsitans*), known locally as the tsetse-fly.[1] This disease occurs in all the domestic animals, but not in man. The permanent source of infection is found in the healthy big game of the 'fly-belt.' *T. equinum* causes the *mal de caderas* of horses in Central and South America. *T. equiperdum* causes the *dourine* of horses in India and Algeria; so far as is known, it is conveyed only by coitus. *T. Theileri* is pathogenic in cattle only; the animals become very anæmic, but usually recover. *T. Gambiense* was the first trypanosome to be found in man, and was discovered by Forde, in 1901, in the blood of a European in the Gambia, who was suffering from an illness supposed to be malarial, but not amenable to quinine. The parasite was subsequently identified as a trypanosome by Dutton. In 1903, Castellani found trypanosomes in the cerebro-spinal fluid of cases of sleeping sickness. Bruce pointed out the connection of parasite and symptoms as cause and effect, and he showed that the disease was transmitted by a certain tsetse-fly—the *Glossina palpalis*. It has since been shown that the trypanosome in question is *T. Gambiense*.

Most of the known trypanosomes of mammals are conveyed from host to host by biting flies.

Trypanosomiasis in Man.—In an endemic region, such as Uganda, a considerable proportion of the apparently healthy natives have trypanosomes in their blood, and it is at least possible that in some instances there may be complete tolerance. In any case, a long period, extending to months and even years, may elapse after infection before the onset of symptoms. These include recurring attacks of fever (*trypanosoma* or *Gambia fever*), loss of flesh and strength, anæmia, œdema, an erythematous eruption, enlargement of the spleen, enlargement of the lymph glands, and sometimes mania or melancholia. It is possible that some cases may undergo arrest in an early stage, but this is not certain. The illness may last for years, with remissions and exacerbations, and may be terminated by some fatal intercurrent

[1] The native term *tse-tse* is derived from the loud buzzing sound made by the insect as it flies.

affection such as pneumonia, or may pass into its hopeless terminal phase, sleeping sickness.

Sleeping Sickness (*African* or *Negro Lethargy*) was known a century ago among the negro slaves, both on the West Coast of Africa and after transportation to the West Indies. It was observed in the latter place that they were attacked from five to eight years after leaving Africa, so that during all this period they must have been the subjects of trypanosomiasis. In 1896 sleeping sickness was brought from the Congo to Uganda by the survivors of the Emin Pasha expedition who settled in the province of Busoga on the Victoria Nyanza. In a few years it has killed hundreds of thousands of people in Uganda. The *glossina* was ready to spread the infection, whereas, apparently, in the West Indies there were no suitable biting flies. Manson recorded the first case of sleeping sickness in a European in December, 1903; but it is now believed that blacks and whites, as well as both sexes and all ages, are alike susceptible. The onset of this terminal stage is characterised at first by slight symptoms, such as lack of facial expression, and a slow, monotonous style of speech. Tremors of the tongue, lips, and hands, and a certain amount of ataxy are also recognisable. The pulse is accelerated. These symptoms gradually become worse, and in the course of from six months to a year the patient becomes bedridden. The lethargy continues to increase till death takes place. The duration of sleeping sickness varies from several months to as many years.

In addition to the enlargement of the lymph glands, lymphocytosis is present at all stages of trypanosomiasis. The onset of the nervous phenomena of sleeping sickness coincides with the entrance of the trypanosomes into the lymph spaces of the nervous system, and is associated with an increase of the lymphocytes in the cerebro-spinal fluid. In some cases, however, there is a late secondary infection of the meninges with bacteria, and especially with a diplostreptococcus. The parasite may not be easy to find in the blood, unless with the aid of the centrifuge, but is readily got in the juice of the enlarged lymph glands—*e.g.*,

in the posterior cervical region. In sleeping sickness it is present in the cerebro-spinal fluid.

Morbid Anatomy.—Examination after death shows increase of the cerebro-spinal fluid, an accumulation of lymphocytes in the perivascular lymph spaces, a dull, ground-glass appearance of the soft membranes, and flattening of the convolutions. The condition appears to be a chronic meningo-encephalo-myelitis. Multiple ulcers are found in the stomach as a result of the digestion of petechiæ by the gastric juice.

Treatment.—There is no prospect of rooting out trypanosomiasis by active immunisation, because it appears that in animals which are rendered immune the trypanosomes do not die out. They simply cease to cause symptoms, and accordingly the animals remain as sources of infection for others of their kind. When arsenic is given to human beings infected with *T. Gambiense*, it destroys some of the parasites, and it is possible that these act as immunising agents. The drug is therefore indicated in the early stages. Trypanroth is said to have given better results than arsenic, and a combination of the two is spoken of as better still. Malachite green has been found destructive to trypanosomes in the blood of experimental animals. These remedies, however, are still on their trial, and the agent has yet to be found which will act upon the trypanosome as quinine acts upon the parasite of malaria.

43. PIROPLASMOSIS.

Belonging to the class *sporozoa* of the *protozoa* are the *hæmocytozoa*, or *endoglobular hæmatozoa*. The *hæmocytozoa* are divided by Laveran into three genera, viz. (1) *Hæmamœba ;* (2) *piroplasma ;* and (3) *hæmogregarina*.

1. *Hæmamœba* includes the malarial parasites of man and the corresponding blood parasites of birds, monkeys, bats, cattle, and tortoises.

2. *Piroplasma* includes several species. *P. bovis* (*v. bigeminum*) is the parasite of red water or Texas fever in cattle. *P. parvum* is the cause of African East Coast fever (Rhodesian

red water), a very fatal disease of cattle in South Africa. *P. equi* causes the biliary fever of horses, mules, and donkeys. *P. canis* causes the malignant jaundice of dogs. The name *P. hominis* has been given to a parasite supposed to be the cause of a disease known as *tick fever* or *spotted fever* (*piroplasmosis hominis*), which has been described in recent years as occurring in Montana, Nevada, and other of the United States. The parasite is transmitted by a tick. Confirmatory evidence, however, is required before these views can be accepted as completely established. The invasion of the disease takes place in from three to ten days after the bite, with chilliness, nausea, headache, constipation, epistaxis, and slight bronchitis. An eruption of bright red macules usually develops about the third day, and occasionally becomes petechial. Albuminuria is always present. A lysis begins about the twelfth day. The mortality is about 70 per cent. Quinine (15 grains hypodermically every six hours) is said to act as a specific.

Several, if not all, of the piroplasma infections mentioned are transmitted by ticks.

3. *Hæmogregarina* occurs in fish, amphibians, reptiles, and mammals. No species is known to be pathogenic.

44. ACTINOMYCOSIS.

Definition.—A chronic infective disease, due to a specific organism, and attended by the formation of granulomata and chronic abscesses.

Etiology.—The organism is an actinomyces or ray-fungus, the *Streptothrix actinomyces*, which belongs to a group intermediate between the bacteria and the moulds.[1] In the pus from the lesions little yellowish masses can be recognised, which consist largely of mycelium. The spores develop on special fruit-bearing hyphæ, and then grow into threads, which branch dichotomously. These threads form a closely-felted radiating mycelium, which constitutes one of the little masses seen in the discharge. In cattle especi-

[1] It is now known that several species of actinomyces are pathogenic in man.

ally, but not so constantly in man, the ends of some of the filaments become club-shaped. This is doubtless a degenerative change, since in cattle the enlarged ends often become calcified, and in artificial cultures the appearance is observed only in old growths.

Actinomycosis is chiefly met with in cattle (in which it is the cause of 'wooden tongue' and 'lumpy jaw'), but it also occurs in pigs. It is almost never transmitted directly from one animal to another. The fungus is common on cereals such as barley, and is probably transferred to man either by the chewing of raw grain, etc., or by the inhalation of dust in such operations as threshing. It is more common in men than in women.

Morbid Anatomy.—In the first place, the specific lesion contains epithelioid cells, small round cells, and sometimes giant cells, together with the organism. Around this granuloma there is a great development of fibrous tissue, and the condition, especially on the face, may readily be mistaken for sarcoma. Secondly, softening, suppuration, and sinus-formation may ultimately occur.

The disease may involve almost any organ in the body, but is specially common in the mouth and its neighbourhood. After this come the alimentary and respiratory tracts, with the peritoneum and pleuræ and the mediastinum and vertebræ. The lymph glands are not readily involved, which is a point of some importance in the diagnosis from tuberculosis. The disease spreads chiefly in a slow manner by infiltrating contiguous parts, but widespread dissemination is not uncommon. The latter is believed to take place by way of the bloodvessels, in which case the infection amounts to a chronic pyæmia.

In the case of the *face*, *jaw*, and *neck* there is a chronic hard swelling, which after a time breaks down and discharges pus. The characteristic granules may be recognisable in the discharge. The disease often spreads in the tissues of the face and neck, and if the periosteum should be attacked, necrosis of bone may ensue. The virus is believed to enter by the buccal mucous membrane.

The *lungs* and *pleuræ* may be infected by way of the

bronchi or from the pharynx or gullet. The symptoms induced resemble those of chronic interstitial pneumonia or of fœtid bronchitis. Sinuses sometimes penetrate through the chest-wall, and the ribs may be damaged. The lesion is usually at the base, and not at the apex ; hæmoptysis is rare ; and the temperature and general health are but little disturbed—points of distinction from phthisis. Unless the specific fungus can be found in the sputum, certainty in diagnosis may be impossible. Death commonly results in about a year.

When actinomycosis involves the abdomen, the usual starting-place is the *intestine*, at or near the cæcum. The mucous membrane may be ulcerated. The peritoneum is thickened and adherent, and chronic abscesses may develop.

The *liver* is sometimes affected, no doubt secondarily to disease of the bowel or of some other part. Solitary or multiple abscesses may be produced, or there may be a diffuse infiltration. The organ may be much enlarged, and cancer, or occasionally tropical abscess, may be simulated.

The *skin* is sometimes attacked as a consequence of local inoculation. Tumours develop and suppurate, and give rise to chronic ulceration.

The *brain* is occasionally the seat of actinomycotic abscess. The symptoms may point to tumour.

Diagnosis.—The only certain test is the presence of the little sulphur-yellow masses, with the characteristic fungus, in the sputum, pus, etc.

Prognosis.—This depends on the possibility of completely extirpating the lesion by surgical measures.

Treatment.—The only satisfactory treatment is excision of the lesions. Failing this, potassium iodide should be administered in doses of from 40 to 60 grains daily, since by means of this drug very encouraging results have been obtained.

45. MILIARY FEVER (Sweating Sickness).

Definition.—An acute epidemic infection, characterised by fever, profuse sweating, and an eruption.

Etiology.—The cause of the disease is unknown. It was much more common several centuries ago than it is now, and it proved very fatal in England in the fifteenth and sixteenth centuries. In more recent times epidemics have occurred in France, Austria, and other European countries, but some of these outbreaks have been of very short duration, and confined to a very small area, such as a single town. Like influenza, the disease may attack a large number of people in a short space of time.

Symptoms.—At the outset there are the usual early symptoms of a fever, and these are followed after a day or so, or perhaps with scarcely any interval, by a drenching perspiration which continues throughout the illness. Constipation, a sense of oppression, and high fever are commonly present. On the third day there is an eruption of red miliary papules, some of which develop a white top. Numerous clear vesicles (sudamina) also appear. The total duration of the attack is nine or ten days. It is associated with severe emaciation, and is often followed by a relapse. Pregnant women almost always abort.

Prognosis.—The mortality varies from 5 to 25 per cent. Fatal cases are characterised by severe nervous symptoms, such as hyperpyrexia, delirium, convulsions, and coma. Death may actually occur within a few hours after the onset.

Treatment.—Cold or tepid sponging and copious draughts of water are the principal measures indicated.

SECTION II

CONSTITUTIONAL DISEASES

1. GOUT (Podagra).

Definition.—A nutritional disease associated with an excess of uric acid in the system, and characterised by the deposition of sodium urate in the tissues of joints and elsewhere, by attacks of acute arthritis, and frequently by disorders of important viscera.

Etiology.—Gout occurs chiefly in middle and advanced life, and is most common in males. Women do not usually suffer till after the menopause. In a large proportion of cases the tendency to the disease is inherited, and females sometimes transmit it without themselves suffering. If the hereditary tendency is very strong, the disease may appear in early life. A gouty individual may transmit the disease through several generations, in spite of every care being taken by his descendants to avert it.

Abundance of alcoholic drinks (especially malt liquors and strong or sweet wines), abundance of nitrogenous foods, and deficiency of physical exercise are three causes of great importance. They are capable of developing gout in those who have no hereditary predisposition, as well as of aggravating it in those who have an inherited tendency.

Chronic lead-poisoning, as in painters, is another well-known cause (saturnine or lead gout). Even the medicinal administration of lead to a gouty person is apt to bring on an attack of acute gout.

An acute attack may be excited by unusual excess in drink or food, or by some depressing influence, such as

worry, emotion, or injury; but where the tendency is strong there may be no obvious exciting cause. Gout is much more common in England than in Scotland or Ireland.

There is great diversity of opinion as to the part played by uric acid in gout, and the account which follows is to be regarded as a provisional hypothesis based on the best scientific opinions. It is well to start with some researches made by the late Sir William Roberts on the salts of that acid. Thus uric acid, $H_2\bar{U}$ (where $\bar{U}$ is $C_5H_2N_4O_3$), is a bibasic acid, which never exists in the blood in a free state. It forms three kinds of salts, namely:

1. The neutral urate, $M_2\bar{U}$, where M is a monatomic metal such as sodium. This does not exist in the living body.

2. The biurate, $MH\bar{U}$, where only half of the replaceable hydrogen is replaced by metal. This is only encountered pathologically in the form of gouty concretions (which consist of sodium biurate).

3. The quadriurate, $MH\bar{U}$, $H_2\bar{U}$, where only a quarter of the replaceable hydrogen is replaced by metal. This exists in the form of sodium quadriurate in the blood of gouty subjects.[1] As the quadriurate accumulates in the blood, which contains sodium carbonate, it tends to take up an additional atom of the base, and is thus converted into the more stable but less soluble biurate; and if the latter is present in any quantity, it is apt to be precipitated in the crystalline form in tissues where the circulation is sluggish. Whatever harm it may do otherwise, uric acid in the form of biurate of sodium causes the phenomena of gout only as a mechanical irritant of tissues, and not as a chemical poison.

Uric acid and the so-called *purin* or *alloxur bases* belong to the group of *purin bodies*. These all contain the hypothetical compound *purin* (from the Latin, *pus*), $C_5H_4N_4$. They result from the disintegration of the nucleo-proteids, which are abundant in the nuclei of cells. It has been supposed that the purin bases (the most important of which are adenin, $C_5H_4N_4$—NH; guanin, $C_5H_4N_4O$—NH; hypoxan-

[1] In normal urine, uric acid exists in solution only in the form of quadriurate, and the familiar amorphous uratic sediment consists of quadriurates. The urine of birds and reptiles consists wholly of quadriurate.

thin, $C_5H_4N_4O$; and xanthin, $C_5H_4N_4O_2$) are the more immediate products of this disintegration, and that they in turn are in great measure converted into uric acid, $C_5H_4N_4O_3$, by oxidation. The purin bodies found in the urine are *endogenous* when due to tissue metabolism, and *exogenous* when derived from the food. Such meats as sweetbread and liver are rich in nucleated cells, and therefore in nucleo-proteids, so that their use leads to an increase in the exogenous purin bodies of the urine. Caffeine, theobromine, theine, and theophylline are themselves purin bodies. It is to be noted, however, that the uric acid in the urine is not all derived from the endogenous and exogenous nucleo-proteids; and, on the other hand, that the nitrogen of the nucleo-proteids is not all eliminated in the form of purin bodies, since some of it is converted into urea.

The endogenous uric acid of the urine is naturally much increased under conditions characterised by great destruction of cells rich in nucleo-proteids. It is accordingly increased in leucocythæmia and in leucocytosis, no doubt in consequence of excessive degeneration of leucocytes and setting free of nucleo-proteids. In chronic leucocythæmia there may be an increase of the purin bases, though the uric acid is not increased.

The reason for the accumulation of uric acid in the blood of gouty individuals appears to be deficient excretion rather than excessive production, and the deficient excretion is in turn attributable to impairment of the function of the kidneys. There is good reason to believe with Garrod and Luff that uric acid is normally produced in the kidneys, possibly by the combination of urea with glycocine brought from the liver. The uric acid, therefore, manufactured in the kidneys, but not excreted in the gouty subject as it ought to be, is absorbed into the general circulation as sodium quadriurate. Antecedent to the gouty blood state, there would thus appear to be something which impairs the excretory function of the renal epithelium, and this is doubtless how lead-poisoning causes gout.

Various other theories are held. According to one, the liver is at fault. Duckworth holds that gout is due to a functional disorder of the medulla oblongata. According to

Ebstein, the primary lesion is a change in the nutrition of the tissues of the affected joint; this goes on to necrosis, and the biurate is then deposited in the necrosed tissue.

Morbid Anatomy.—Uric acid is present in the blood,[1] and may be recognised by Garrod's test. Some serum obtained directly from the blood or from a blister is placed in a watch-glass and acidulated with acetic acid. A linen fibre is then placed in the fluid, and the latter is covered and left to evaporate in a warm atmosphere. If the gouty condition is present, microscopic examination will then show uric acid crystals adhering to the fibre.

The biurate is deposited in the connective tissues, especially of joints, but also in the ear and other parts. The metatarso-phalangeal joint of the great toe is the most frequent seat of deposit. This takes the form of fine needles arranged in a stellate manner in the superficial layers of the cartilage, but not on the surface. The appearance of an articular cartilage containing this deposit is, as Duckworth remarks, exactly like that which would result from smearing it or splashing it with white paint. The biurate may also be deposited in the other tissues of joints, or in parts at a distance, as in the external ear, tendons, skin, cerebral membranes, and kidneys. The selection of particular tissues by the disease is probably connected with the amount of strain which those tissues habitually undergo. Thus washer-women suffer in their hands. In the acute attack there is arthritis with effusion. In cases of long standing the joints are enlarged, deformed, and stiff, and the urate in the joints, or elsewhere, collects into chalk-like deposits (*tophi*, or chalk-stones), which occasionally ulcerate through the skin.

In addition to being the seat of uratic deposits, as just mentioned, the kidneys are often the seat of chronic interstitial inflammation (gouty kidney); but neither of these conditions is peculiar to gout. The cirrhotic kidney is regularly associated with cardiac hypertrophy, and often with arterial degeneration.

Symptoms.—When gout involves a joint, it is called *regular* or *articular;* when it is present elsewhere, it is called *irregular*

[1] Uric acid is not found in the blood of healthy persons, but is sometimes found in leucocythæmia and severe anæmia.

or *abarticular*. Sometimes the two forms co-exist. When gout suddenly leaves a joint, and gives rise to symptoms connected with an internal organ, it is called *metastatic*, *retrocedent*, or *suppressed* gout. If the attacks rapidly flit from place to place, it is *flying* gout. Articular gout may be either *acute* or *chronic*.

Acute Gout.—Sometimes the gouty paroxysm is preceded by slight symptoms, such as trifling pains in the joints, dyspepsia, and mental irritability. The attack usually sets in in the early morning, the patient being awakened by severe pain in the first metatarso-phalangeal joint, usually of the right side. The pain becomes intense, but, after some hours, abates. The joint swells, and is extremely tender. The skin over it is red and œdematous, and the veins are distended. There is slight fever, and the urine is concentrated. Furring of the tongue, anorexia, thirst, and constipation are other general phenomena of the attack. During the day the patient may be fairly comfortable, but the symptoms recur each night. In this way, unless efficiently treated, the 'fit,' or gouty paroxysm, is prolonged for a week or two. The joint does not suppurate. As the inflammation passes off, well-marked desquamation sets in.

After the early attacks, the affected joints usually recover to all appearance completely. Moreover, the patient often feels much better than he did for a considerable time before the fit. He is now likely to remain free from attacks for a period of months, or even a few years. But after several fits have occurred, the intervals tend to become shorter, more joints are involved, the permanent damage to joints becomes greater, and the recovery in the intervals is less complete. And so the disease merges into chronic gout.

Suppressed Gout.—If the fit is suppressed, articular symptoms may be replaced by gastro-intestinal symptoms, such as pain, vomiting, diarrhœa, and faintness; or by cardiac symptoms, such as pain, palpitation, and dyspnœa; or by cerebral symptoms, such as delirium or apoplexy. It is possible that some of these phenomena are uræmic, and that the cardiac manifestations really constitute an attack of angina pectoris.

Chronic Gout.—If the acute attacks are frequent and pro-

longed, more and more joints suffer, and ultimately serious deformity and immobility may ensue. Tophi develop around the joints, in bursæ, and in the ears. Sometimes the overlying skin gives way—for instance, over the knuckles—and allows the chalk-like material to escape, either in a creamy consistence or in solid particles. Occasionally suppuration takes place around the chalk-stones, and promotes their removal. The chalky material consists of microscopic, needle-like crystals of sodium biurate. The condition of the urine and of the cardiovascular system is often indicative of the gouty kidney. Death may thus result from cerebral hæmorrhage, uræmia, inflammation of serous membranes, etc.

Irregular Gout.—This designation is applied to various non-articular affections met with in persons who, from heredity or from their manner of life, are predisposed to gout. Possibly the inflammatory affections of this class are actually due to deposits of the biurate. Thus there may be catarrh of the respiratory, alimentary, or urinary tract, asthma, angina pectoris, glycosuria, calculus, eczema, psoriasis, pruritus, phlebitis, iritis, meningitis, neuritis, neuralgia, etc.

Diagnosis.—A typical attack of acute gout presents no difficulty. In chronic cases, especially if the foot does not suffer first, there may be some doubt, and tophi should always be searched for.

In *osteo-arthritis* there are no tophi. Changes in the bones take place early. The hands suffer more than the feet. The severe forms usually occur earlier in life than gout, are common in women, and are due to causes different from those of gout.

Rheumatism occurs chiefly in young adults and children. Females often suffer. A history of gout in the family is probably awanting, whilst a rheumatic family tendency may be recognisable. The arthritis involves several joints even in the first attack. It shows no preference for the great toe, but rather for the middle-sized joints. It tends to flit from joint to joint in the course of one attack. The onset is not sudden as a rule. The joints are swollen and occasionally reddened, but not œdematous or covered by distended veins. There are no tophi.

Prognosis.—Occasional acute attacks are not dangerous to life, but the cerebral, cardiovascular, and renal disorders of chronic, irregular, and suppressed gout are often the cause of death in gouty subjects.

Treatment.—Individuals who are in any way predisposed to gout should be specially temperate in eating and drinking. Alcohol should, if possible, be avoided altogether, but if it cannot be dispensed with, it should be taken in the form of whisky or very old brandy, and not as wine or malt liquor. Abundance of fresh air, regular hours, and regular exercise short of fatigue, are important. The diet should include a moderate amount of nitrogenous food. White fish may be taken very freely, and butcher's meat more sparingly, but meat extracts ought to be avoided. Pastries and sweet things in general should also be shunned. Hot water may be taken with advantage on rising and at bedtime. An occasional dose of blue pill at night, followed by an alkaline purge in the morning, may be recommended. Mineral waters are useful chiefly on account of the water itself; if gastric or hepatic complications are present, some of the mineral ingredients may also be of some service. But when patients go to Bath, Buxton, or Aix-les-Bains, they drink the water in a prescribed manner, abjure for the time high living and late hours, and have exercises, massage, baths, etc., to stimulate the emunctories.

In the acute attack the inflamed part should be elevated, and wrapped in cotton-wool, or fomented. A mercurial purge and a ½-drachm of colchicum wine should be given at once. Thereafter 20 minims of the wine, with 1 drachm of potassium citrate, may be given every six or eight hours, but the colchicum should be stopped as soon as the acute pain has been relieved. If colchicum fails, morphine may be necessary. The diet should consist of milk and light farinaceous articles, with plenty of plain water, or mineral water free from sodium.

The treatment of irregular and suppressed gout depends largely upon the organ affected. In addition to dietetic measures, iodide of potassium and bitter tonics may be beneficial.

In chronic gout, the remedies which deserve a trial, in

addition to dietetic and hygienic measures, include colchicum, guaiacum, quinine, arsenic, mineral waters, and baths, with or without a sojourn at one of the watering-places which specially cater for this class of case.

2. CHRONIC RHEUMATISM.

Definition.—A very chronic affection, occurring chiefly at and after middle life, and characterised by stiffness and sometimes swelling of joints, and by pain, which is often aggravated in cold and damp weather.

Etiology.—Quite exceptionally this disease may follow acute or subacute rheumatism. It usually sets in gradually in persons who have reached or passed middle life, and who, through their occupations, are much exposed to cold and wet.

Morbid Anatomy.—There is very little effusion, and the cartilages and bones, even in severe cases, may be scarcely altered. But the capsule, the ligaments, and the sheaths of neighbouring tendons, are thickened, so that movement is impaired. Movement is often associated with a peculiar sound.

Symptoms.—Pain and stiffness are the principal symptoms. There may be swelling, The affected joints may be few or many, and the pain is often aggravated on the approach of cold or wet weather. The pain is a severe aching, and is often relieved by rubbing and by using the limbs. The general health may be unaffected, but prolonged and severe suffering, with loss of sleep, may lead to some deterioration. Degenerative changes may be present coincidently in the valves of the heart.

Diagnosis.—In *osteo-arthritis* the bones and cartilages suffer early.

In *chronic gout*, tophi, and a family history and other evidences of the gouty diathesis may be looked for.

Prognosis.—The disease does not definitely shorten life, but the pain and stiffness may be very obstinate and persistent.

Treatment.—Patients who can afford it should winter in a mild, dry climate. A sufficiency of woollen clothing is very important. It may be necessary to try a number of drugs internally before a suitable one is hit upon. Potassium

iodide, sodium salicylate, guaiacum, sulphur, arsenic, and iron may be tried. Local measures may also be employed—the application of methyl salicylate as a wet dressing, faradism, blistering, painting with iodine, the actual cautery, hot douching, exposure to hot air, and persevering massage. Resort may be had to Bath, Buxton, Strathpeffer, Aix-les-Bains, or some other suitable watering-place. If severe pain is associated with the presence of a well-defined fibrous tissue nodule, the latter may be excised ; or, as Stockman recommends, from 5 to 8 minims of a 1 per cent. watery solution of chromic acid may be injected into its substance.

3. MUSCULAR RHEUMATISM (Myalgia).

Definition.—A painful affection of voluntary muscles and of the fibrous tissues with which they are connected.

Etiology.—The attacks often result from exposure, especially in persons of a rheumatic or gouty tendency. The disease also results from strain of the fibro-muscular structures. One attack predisposes to another. Stiff neck is specially common in young subjects, lumbago in older people.

Morbid Anatomy.—No anatomical changes are known to exist in the affected tissues. The facts, however, point to the disease being an inflammation of the fibrous tissue of muscles. The ends of the sensory nerves of the muscles are situated in that fibrous tissue, and suffer along with it.

Symptoms.—The onset is often sudden, but there may be little or no constitutional disturbance. Pain is the leading symptom, and this is greatly aggravated, and, indeed, may be only experienced when the affected muscles are put into action. There are thus both an attitude and an actual condition of stiffness, resulting from the desire to avoid movement. The usual duration is several days. The following are the most important varieties :

Rheumatic torticollis (stiff neck, acute wry neck) is specially common in young subjects. The sterno-mastoid of one side is chiefly involved, but other muscles on the side of the neck may be painful. It causes a fixed attitude of the head.

Pleurodynia (intercostal myalgia) occurs chiefly in adults,

and often results from severe coughing. An intercostal, pectoral, or serratus muscle may be at fault. There may be very localised tenderness. The suffering is apt to be great, since between breathing and coughing the muscle is not allowed to rest. The respiratory movements are restricted on the affected side, but there is no pleuritic friction.

Lumbago affects the muscles of the small of the back, and, while generally bilateral, is often accompanied by unilateral sciatica ; the inflammation of the fibrous tissue of the muscles extends to the fibrous sheath of the nerve. Intense pain may be caused by an attempt to straighten the back after stooping. The affection occurs chiefly in adults.

In *omodynia* the muscles about the scapula are involved.

Diagnosis.—*Neuralgia* is distinguishable by its paroxysmal and often periodic character, the different etiology, the absence of stiffness, and the slight influence of movement.

Prognosis.—Recovery is almost certain to take place, but renewed exposure may lead to recurrence.

Treatment.—Rest is important, and an alkaline purge and diuretic should be given at the outset. A vapour bath may be helpful. Severe pain may necessitate an injection of morphine. In pleurodynia, the affected side of the chest should be strapped. In lumbago, fomentations are very useful. Another valuable procedure is acupuncture, long needles being inserted into the painful muscles, and withdrawn after ten minutes. Anodyne liniments or the positive pole of the galvanic current may be used for sedative purposes, and blisters or the cautery for counter-irritation. Strychnine, quinine, potassium iodide, guaiacum, and sulphur may be tried in chronic cases. Flannels should be worn habitually, and draughty water-closets must be avoided.

4. OSTEO-ARTHRITIS

(Arthritis Nodosa. Arthritis Deformans. Rheumatoid Arthritis. Chronic Rheumatic Arthritis. Rheumatic Gout).

Definition.—A chronic disease characterised by changes in the tissues of joints, and leading to deformity.

Etiology.—The disease, as seen in hospitals, generally

begins in adults before the age of fifty. Women suffer more than men, especially from the severe types. Among the causes are debilitating influences of all kinds, such as cold, damp, menstrual and other uterine disorders, lack of food and clothing, and anxiety. Joints that have been injured and joints which have been the seat of ordinary rheumatism are predisposed to osteo-arthritis. The joints may have an inherited susceptibility.

The pathology is obscure. The idea that the disease is a combination of rheumatism and gout ('rheumatic gout') is practically obsolete. The theory that it is a trophoneurosis receives support from the similarity of the lesions in the joints, muscles, and skin to those of locomotor ataxy and syringomyelia, and from the symmetry and progressive nature of the disease; but anatomical evidence is still awanting. Another view is that it is a chronic infection, and a micro-organism has been found in the joints by Bannatyne, Wohlmann, and other observers; but here, too, the evidence is not conclusive.

Morbid Anatomy.—The joints are enlarged and often contain fluid. The changes begin in the cartilages and synovial membranes. The synovial fringes enlarge, and cartilage may develop in them, and may even become partly ossified. The articular cartilages become soft and velvety, and are worn away by the friction of the opposed surfaces. The ends of the bones thus exposed become dense and polished (eburnated). At the edges, where there is but little pressure, hypertrophy of the cartilage and other tissues takes place, and this leads to calcification and ossification, so that the margin is lipped. The bony outgrowths which develop at the margins of the joints are called 'osteophytes.' The ligaments may be much thickened, and the result of all the changes is to greatly diminish or entirely abolish the mobility of the joint. Such ankylosis, however, except in the case of the spinal column, is not due to actual bony union.

The muscles which act upon the joints undergo wasting. Inflammation has sometimes been found in neighbouring nerves. Subcutaneous fibrous-tissue nodules are occasionally found. The heart rarely suffers in this disease.

Symptoms.—Several clinical types are recognised:

1. The *chronic polyarticular* form is the most important. The smaller joints (metacarpo-phalangeal, interphalangeal, etc.) tend to suffer first and most, but the middle-sized and large joints are often involved also. The joints of the spine and lower jaw may suffer. Pain, tenderness, impairment of mobility, and enlargement of the ends of the bones, often with effusion in the joints and slight reddening of the skin, are the principal local phenomena. Grating is sometimes felt on passive movement of the joints. The disease is roughly symmetrical. There is often deflection of the hand to the ulnar side. The metacarpo-phalangeal joints are flexed, and those of the second and third digits are particularly apt to show early and persistent swelling. The interphalangeal joints may be flexed, extended, or distorted in an irregular fashion. The muscles waste, and there may be distinct evidence of neuritis. There may be slight pyrexia and general debility. In severe and advanced cases, debility and anæmia are well marked, and considerable pigmentation of the skin may be present. Under treatment many of the cases cease to progress, and considerable improvement may take place.

2. The *acute polyarticular* form is much less common. No enlargement of the bones is recognisable, but there is fluid effusion with swelling of the synovial membrane and rapid destruction of cartilage. Fever is present, and the case may thus for a time resemble ordinary rheumatism. After some weeks the disease becomes stationary, or merges into the chronic type.

3. The *localised* form is often monarticular, and affects especially the hip (*malum coxæ senile*) or shoulder in elderly persons. It may, however, affect a portion, or the whole, of the spinal column. The condition may follow an injury. Pain, muscular wasting, impaired mobility, and grating are the principal signs. The hard tissues of the joint may be greatly altered, but a considerable amount of movement is usually retained.

There are two varieties of *progressive ankylosis of the spine*, one or both of which may perhaps belong to the localised type of osteo-arthritis. (1) The Von Bechterew type (*spondylitis deformans*) is characterised by rigidity of the spine, with root-pains and degeneration of the posterior roots and

columns. (2) The Strümpell-Marie type (*spondylose rhizomélique*) is characterised by rigidity of the spine, with disease of the large joints such as the shoulder and hip, but without marked nervous symptoms. I have met with a case where the ankylosis of joints was almost universal.[1]

4. *Heberden's nodes* (*digitorum nodi*) are the bony outgrowths found in the fingers, especially at the distal interphalangeal joints, and particularly in elderly women. At first there may be some pain, but, as a rule, the condition comes under the notice of the physician accidentally. The patient, fortunately, is not likely to suffer from the severe types of the disease.

5. A special type has been described by Still as occurring in children before the second dentition. There is swelling with great stiffness of the joints, and the affection usually begins in the knees, wrists, and cervical spine. The swelling is connected with the periarticular tissues, and is not due to changes in the bones or cartilages, so that there is no grating. The condition is painless. There may be fever, with enlargement of the related lymph glands and of the spleen, and there is often profuse sweating. It has been supposed that this type is of tubercular origin.

Diagnosis.—In *acute rheumatism* there is no hypertrophy of the bones or ligaments, the arthritis shifts from place to place, visceral lesions are often present, and the history may reveal a rheumatic tendency.

In *chronic rheumatism* there is little or no swelling, and the bones and cartilages are altered but slightly, if at all. The general health may be good. The disease occurs principally at and after middle life, whereas the painful forms of osteo-arthritis, with the exception of the monarticular variety, are met with chiefly before middle age.

In *chronic gout* there may be tophi, or a history of acute attacks.

Where the shoulder-joint is suspected of being the seat of osteo-arthritis, care should be taken to exclude *trophic conditions* of the joint and its muscles, resulting from lesion of a nerve.

Charcot's joint disease, a trophic lesion which sometimes

[1] *British Medical Journal*, May 25, 1901.

complicates locomotor ataxy, is characterised rather by an increase than by a diminution of mobility. There may be considerable effusion without pain in the affected knee or other articulation. The distinctive symptoms of locomotor ataxy are likely to be present.

Prognosis.—There is no danger to life except in those rare instances where the bony wall of the chest is fixed, and where, accordingly, any catarrhal condition of the respiratory tract will involve considerable risk. Pain may be at first very severe, and for a considerable time troublesome. Mobility is greatly impaired in bad cases, but much benefit may be looked for from appropriate treatment.

Treatment.—It should be remembered that the disease is one of debility, and that depressing modes of treatment are objectionable. In acute and febrile cases Dover's powder or some other opiate may be given to procure sleep, and one or two moderate doses of sodium salicylate may sometimes be given daily with advantage. In less severe cases the same remedies should be employed, with the addition of arsenic, and the patient should have an abundance of nourishing food, with cod-liver oil, and perhaps iodide of iron or of potassium. Carbonate of guaiacol is another useful drug.

Local treatment is very important. Fomentations should be applied to relieve severe pain. Methyl salicylate is another excellent local anodyne. Joints that are not very painful should be immersed for ten minutes in hot water and then passively moved through as large an angle as the patient can tolerate ; this should be repeated once or twice daily. When pain is slight, friction or continuous elastic pressure may be employed. Small blisters may be applied from time to time. If severe pain persists obstinately, much good may be obtained from the use of Corrigan's button. Flannel clothing should always be worn next the skin. A warm, dry climate is desirable. Patients who can afford it might do well to resort early to Bath, Buxton, or some other of the watering-places which have a reputation in the treatment of osteo-arthritis ; but very encouraging results may be obtained by persevering treatment at home or in hospital, with the aid of a good nurse.

5. RICKETS (Rhachitis).

Definition.—A disorder of the general nutrition occurring in infants, and characterised especially by changes in the bones.

Etiology.—The usual age of onset is between six months and eighteen months.[1] Rickets is specially common among the poor of large cities, because of improper feeding, impure air, and insufficient sunlight. Hand-fed children are apt to get proprietary and other foods containing an excess of starchy matter and a deficiency of fats and proteids, and they suffer more than breast-fed infants. Unduly prolonged lactation and suckling of the child during pregnancy may be contributing causes. The disease is occasionally met with among the children of the rich. It is not inherited. A family tendency to tubercle is believed to be antagonistic to the development of rickets.

Morbid Anatomy.—The most marked changes are at the growing ends of the long bones. Ossification from cartilage at the junction of shaft and epiphysis is delayed and irregular. The bluish zone of multiplying cartilage-cells is greatly swollen, and no longer bounded by well-marked transverse lines. The yellow zone of calcification of cartilage is also irregular, and portions of yellow may appear in the blue zone. Bloodvessels may appear in the cartilage. The osteoblasts under the periosteum of the shaft also increase ; but here, too, bone is imperfectly formed, and as the medullary cavity continues to grow at the usual rate, the proportion of osseous matter in the thickness of the shaft is diminished. The softening is not due to morbid absorption of earthy matter from bone already ossified, but to delay in the ossification of new bone. Thus the ends of the bones are enlarged and softened, and the shafts are thickened and too readily bent. It has been supposed that the immediate cause of these changes is local hyperæmia.

[1] A rare disease, characterised by enlargement of the ends of the bones and bending of the limbs, has been described as occurring in older children, and has been named *late rickets*, but its nature is doubtful.

The liver, spleen, and lymphatic glands may be enlarged. The muscles are pale and flabby.

Pathology.—Different theories of rickets have been proposed. The view that it is due to deficiency of lime in the food has not proved satisfactory. Another theory is that it is an infection, and another that it depends upon atrophy of the thymus.

Symptoms.—The onset is insidious.[1] A very characteristic combination of three symptoms was pointed out by Sir William Jenner. First, the child sweats freely about the head; secondly, it throws off the bedclothes, whether the atmosphere is hot or cold; and, thirdly, it cries when it is handled, probably on account of a general tenderness all over. Fever is slight or absent, unless a complication is present. There may be considerable muscular weakness. Numerous changes take place in the bones. The anterior ends of the bony ribs are enlarged, constituting the 'rhachitic rosary.' In inspiration the anterior portions of the softened bony ribs tend to yield, and thus is produced the 'rickety chest,' which is characterised by a shallow vertical groove on each side. Rickets in combination with an impediment to inspiration evolves the 'pigeon-breast' and the transverse depression known as 'Harrison's sulcus.' The spine, from the upper dorsal to the sacral region, shows a single curve with the convexity backwards, the result of muscular and ligamentous weakness. Changes in the shape of the pelvis may cause serious trouble later on in connection with parturition. The face is small in comparison with the head. The teeth appear late and decay early. The frontal and parietal eminences are unduly prominent. The closing of the anterior fontanelle may be greatly delayed. The cranium in the parieto-occipital region may be thin and parchment-like ('cranio-tabes,' a condition met with also in inherited syphilis). The lower ends of the forearm bones are enlarged, and occasionally the upper limbs are much deformed through yielding when the child creeps about. The ends of the long bones of the

[1] The disease formerly called *acute rickets* is now recognised as infantile scurvy. (See Scurvy.)

lower limbs are also enlarged, especially at the ankle. The acquirement of walking may be delayed, and if the child walks, bow-leg or knock-knee will develop. 'Green-stick fracture' of the soft bones may occur. The laxity of the ligaments accounts for the child being 'double-jointed.'

The abdomen is large, owing in part to flatulent distension; but the body as a whole, including the limbs, is stunted in its growth, and the child is often 'backward' in mind also. Sometimes there is enlargement of the spleen or liver. Rickets predisposes strongly to the convulsive diseases of infancy (general convulsions, laryngismus stridulus, and tetany).

Diagnosis. — This depends upon Jenner's triad of symptoms, on the bony deformities, the late teething, the tendency to convulsive disorders, and the conditions under which the child has been reared.

Prognosis.—Rickets is not fatal directly, but complications, such as broncho-pneumonia and diarrhœa, may cause death. Respiratory diseases are rendered more formidable by the yielding condition of the chest-wall.

Occasionally the deformities persist through life, and thus a deformed pelvis may obstruct labour. Severe bow-leg or knock-knee will interfere with walking, and a deformed chest will handicap the individual if he should suffer from severe bronchitis. Sometimes the convulsions of infancy return at puberty, and continue to recur as ordinary epilepsy.

Treatment.—The child should be well clad and kept as much as possible in fresh air and sunlight. It should have a warm bath daily, but chill must be guarded against. The ventilation of the sleeping-room should be attended to.

If the child has been fed at the breast, it ought to be weaned, and to be nourished chiefly on milk diluted with barley-water or lime-water. Occasionally the yolk of an egg may be beaten up in the milk. At the end of the first year beef-juice, beef-gravy, and custard may be given in small quantities. Farinaceous matter must be added to the dietary with the utmost circumspection, lest it cause indigestion and abdominal pain. A dose of castor oil or of rhubarb and soda may now and then be given with ad-

vantage, if there is any evidence that the intestinal contents are causing irritation. Equal parts of cod-liver oil and chemical food make an excellent combination, of which a teaspoonful or less may be given three times a day. Another remedy is phosphorus ($\frac{1}{200}$ to $\frac{1}{120}$ grain), which may be given twice or thrice daily in a sugar-coated pill, or dissolved in olive oil. To avoid deformities as far as possible the child should be prevented from walking. This may be done by applying to the legs light splints which project beyond the feet, and which may be removed at bedtime.

6. MOLLITIES OSSIUM

(Osteomalacia. Malacosteon).

Definition.—A disease characterised by progressive softening of the bones and leading to serious deformity.

Etiology.—The disease is rare, and much rarer in Britain than in certain parts of the Continent. It occurs chiefly among the poor. It is almost confined to adult life, and is many times more frequent in women than in men. The symptoms are often first noticed in pregnancy, and they may cease after parturition, to make progress again in each succeeding pregnancy. The disease attacks chiefly multiparæ. Excess of lactic acid in the bone-marrow has been suggested as a cause, but the true nature of the condition is still a mystery.

Morbid Anatomy.—The bony trabeculæ become decalcified, and soon afterwards the lacunæ with their corpuscles disappear, and are replaced by a soft vascular gelatinous tissue, which may have already replaced the marrow. Hæmorrhages often occur in this new tissue. The process begins in the medulla, and gradually extends towards the surface of the bone. The periosteum, however, remains unaltered. Bone in this condition may be cut with a knife.

Symptoms.—At an early stage there may be pains about the pelvis and limbs, and difficulty in walking. Under the weight of the body the pelvis becomes greatly deformed.

The sacral promontory and the acetabula are approximated to one another, so that the symphysis projects forwards like a beak. A well-marked vertical groove may develop on each side of the thorax through the pressure of the arms upon the ribs. Multiple fractures as well as bendings may occur. Frequently the disease is not recognised till the accoucheur makes a pelvic examination. The lime salts absorbed from the bones are eliminated in the urine and fæces.

Diagnosis.—This depends on the age and sex of the patient, the early affection of the pelvis, and the marked softness of the bones involved.

Prognosis.—Occasionally the disease undergoes arrest, but it is generally progressive, and the patient dies after some years, perhaps from an intercurrent respiratory affection, or as the result of an operation undertaken to effect delivery.

Treatment. — Careful attention should be paid to food and hygiene. Beneficial results have been reported from oöphorectomy and from the internal use of bone-marrow and phosphorus.

7. FRAGILITAS OSSIUM.

Definition. — A condition characterised by abnormal brittleness of the bones of primary origin.

Etiology.—The tendency to this idiopathic brittleness may be inherited and run in families, but otherwise practically nothing has been learned as to its cause, either from morbid anatomy or in other ways.

Symptoms.—The first fracture generally occurs in early life, as a result, perhaps, of the most trifling cause. The bone is not soft, the fracture is not greenstick, and, as a rule, it unites readily. The general health is unaffected. After many fractures have been sustained, there may obviously be some deformity.

8. DIABETES MELLITUS.

Definition.—A disorder of nutrition characterised by persistent and well-marked glycosuria, and often accompanied by polyuria and emaciation.

Etiology.—Diabetes ('the gout of the carbohydrate-eater') may occur at any age, but is specially common between thirty and sixty. It is more common in males than in females. There is sometimes a hereditary or family tendency to the disease. Thus, a boy who recovered in my wards (for the time, at least) was the fifth child of the family to suffer. Two brothers and two sisters had already died of diabetes. The disease is occasionally met with in husband and wife. The rich suffer more than the poor, and the special liability of the Jewish race is well known. Other favouring conditions are obesity, gout, excess in food and drink, a sedentary life, city life, hard brain-work, anxiety, infectious fevers, and lesions of the nervous system. Mental shock, with or without injury to the head, abdomen, or some other part of the body, may be followed by diabetes in the course of weeks or months. Malaria and syphilis have also been regarded as causes.

Pathology and Morbid Anatomy.—Two opposing theories are held, which are based on different views as to the so-called glycogenic function of the liver.

1. On the one hand, Claude Bernard taught that the carbohydrates of food are stored up in the liver in the form of glycogen, and that the latter is reconverted into sugar and discharged into the blood as required for the needs of the organism. Those who follow Bernard, therefore, suppose that in diabetes the excess of sugar in the blood, and consequently its appearance in the urine, are due to increased production of sugar by the liver, or to diminished consumption by the tissues, or to both causes. In severe cases sugar may also originate from the tissues.

2. On the other hand, according to Pavy, any change in the proportion of sugar in the blood is speedily reflected in the urine. The blood, like other parts of the body, contains normally a very minute quantity of sugar, and by modern delicate tests, though not by those ordinarily used in clinical work, sugar can be recognised in the urine. Pavy holds that, under normal conditions, carbohydrate is prevented from reaching the blood by being transformed by the intestinal villi and the liver, partly into fat, and

partly into a proteid which has the chemical constitution of a glucoside. He holds, therefore, that in simple glycosuria there is lessened activity, relatively or absolutely, of the sugar-transforming mechanism, which thus allows some of the carbohydrate to pass. In ordinary diabetes, this deterioration is more serious; whilst in the acute form seen in young adults, not only is there a breakdown of this mechanism, but the proteid of the tissues is split up, probably by a pathologically-developed ferment in the blood, and this proteid (being a glucoside) yields glucose as one product of its decomposition.

Pavy regards diabetes as a disease of the nervous system operating through the vasomotor system.

In half or more of the cases, changes of various kinds are found in the *pancreas*. Moreover, complete extirpation of the pancreas in dogs is followed by diabetes. But if a portion of the pancreas remains in the body, either in its original situation or after transplantation, diabetes does not occur, so that probably this organ produces an internal secretion which decomposes sugar. This, however, does not explain all cases of diabetes, since in a considerable proportion the pancreas has been found normal even under microscopic examination. The part of the pancreas which is connected with diabetes is the islands of Langerhans. These may be looked upon as constituting a ductless gland, and are the source of the internal or glycolytic secretion of the pancreas. When they are destroyed by malignant disease involving the whole gland, or by the interacinar type of chronic interstitial pancreatitis, diabetes results. But in the interlobular type of chronic interstitial pancreatitis the islands of Langerhans escape, and there is no glycosuria. Glycosuria may be regarded as a relatively uncommon symptom of pancreatic disease.[1]

In the *liver* there is no constant lesion, but the organ is often enlarged and fatty. There is no constant change in the *nervous system*, though in some cases intracranial lesions, such as tumour, softening, or hæmorrhage, have

[1] The association of diabetes and pancreatic disease was first pointed out by Cawley in 1788.

been found, and these may have caused the diabetes. Multiple neuritis and degeneration in the posterior columns of the spinal cord may also be present. The *arteries* are often atheromatous. The *blood* contains an excess of sugar and sometimes of fat (lipæmia). Glycogen, which may be present in the plasma of normal blood, is found in diabetes in the polymorphonuclear leucocytes. Tuberculosis of the *lungs* is common, and pneumonia, bronchopneumonia, and gangrene are also observed. In the *kidneys* there may be some fatty change, and there is sometimes hyaline degeneration of the tubular epithelium. The *bladder* is occasionally hypertrophied. Charles Workman holds that the most constant change is hypertrophy of the whole thickness of the *duodenal mucous membrane*.

Symptoms.—The onset, though occasionally acute, is usually gradual. The principal symptoms are increase in the quantity of urine and in the frequency of micturition, thirst, hunger, and loss of flesh and strength. Apart from complications, the skin is usually dry, and the temperature normal or subnormal. The tongue is often abnormally red, and a sweet taste may be present in the mouth. Constipation may be obstinate, though diarrhœa is sometimes present, perhaps owing to the character of the antidiabetic diet. In elderly people diabetes is often of a mild type, and may affect the general health very little.

The *urine* is pale, acid, and sweet-smelling. In cases of moderate severity, the quantity may be 7 to 15 pints, with a specific gravity of 1035 to 1045, and a daily output of 15 to 25 ounces of sugar, which is present in the urine to the extent of from 8 to 12 per cent. The specific gravity, however, may be low, and the quantity but little increased, especially in the mild disease of elderly persons. Febrile attacks and other intercurrent illnesses often cause the sugar to diminish greatly or to disappear for the time being; and, unless in the most severe cases, the output of sugar is much influenced by diet. The sugar is dextrose, $C_6H_{12}O_6$.

Various *tests for sugar* are employed; several of these depend upon the power of grape-sugar to reduce metallic salts in alkaline solution.

1. *Fehling's Test.*—Two solutions are made and are mixed in equal quantities immediately before use. The first contains copper sulphate, sulphuric acid, and distilled water. The second contains sodium potassium tartrate, sodium hydroxide, and distilled water. Equal volumes of these make Fehling's solution. To examine for sugar, a drachm of this solution is put in a test-tube, and boiled to test its purity. If the fluid remains unaltered, the suspected urine is then added in small quantities at a time, the mixture being raised to the boiling-point after each addition. If sugar is present in any quantity, a red (anhydrous) or a yellow (hydrated) precipitate of the suboxide of copper will appear. If the sugar is less than ½ per cent. of the urine, precipitation takes place more slowly, and the change in colour is to olive-green instead of to red. Boiling should not be prolonged, and the urine in the test-tube should never be in excess of the Fehling's solution; otherwise fallacies may arise. For instance, with prolonged boiling the copper salt may be reduced by uric acid, or by glycuronic acid, which may be present in the urine.

2. *Trommer's Test.*—To the urine in a test-tube are added a few drops of a saturated solution of copper sulphate. Then liquor potassæ is added drop by drop, giving a bluish-white precipitate (hydrated peroxide of copper). More liquor potassæ is added till the precipitate dissolves, and a deep blue solution of cupric oxide results. On heating the mixture, the anhydrous (red) or the hydrated (yellow) suboxide of copper is precipitated if sugar be present.

3. *Böttger's Bismuth Test.*—The urine is mixed with an equal quantity of a saturated solution of sodium carbonate; a few grains of nitrate of bismuth are added, and the mixture is boiled. In the presence of sugar the white nitrate is reduced to the metallic state, becoming black. The test is not satisfactory.

4. *Moore's Liquor Potassæ Test.*—Equal parts of the urine and liquor potassæ are mixed and boiled. If sugar is present, a dark-brown colour is developed, and an odour of burnt sugar becomes perceptible. The test is not delicate.

5. *Johnson's Picric Acid Test.*—To a saturated solution

of picric acid some liquor potassæ is added. The mixture is boiled and then added to an excess of urine. If sugar is present, a deep red colour is produced.

6. *Fermentation Test.*—A test-tube is filled with the suspected urine, and a small fragment of yeast is added, the tube being then inverted over a dish containing urine from the same source. If sugar is present, it is decomposed by the yeast into alcohol and carbon dioxide, and this gas, accumulating in the tube, gradually expels the urine. The test is reliable, though not very delicate.

7. *Phenyl-hydrazin Test.*—A test-tube is filled to the depth of ½ an inch with hydrochlorate of phenyl-hydrazin, and the same amount of sodium acetate is added. The tube is then half filled with the suspected urine, and the mixture is boiled for several minutes, and then allowed to stand for from twenty minutes to several hours. If sugar is present, a yellow deposit takes place, consisting of sheaves of bright yellow needle-like crystals of phenyl-glucosazone. This is a very valuable test.

8. The *polariscope* is also available as a test for the presence of dextrose in the urine.

Quantitative Tests.—1. *Fehling's Solution* is the best. A standard solution (different from that described above as used for qualitative work) is prepared so that 10 c.c. of the solution will be decomposed by 0·05 gramme glucose. In practice 10 c.c of the standard solution are put in a porcelain capsule over a spirit lamp, and then diluted with an equal volume of water, and boiled. Obviously the solution in the capsule will be completely reduced—so that its blue colour is lost—as soon as 0·05 gramme sugar has been added to it when boiling. The urine to be examined is diluted with water to 1 in 10, and the diluted fluid is then put into a burette which is graduated from above downwards. The burette is filled up to zero, and fixed over the boiling Fehling's solution. The diluted urine is then allowed to drop slowly into the capsule, and as soon as the blue colour in the latter is discharged, the flow of urine is stopped. The diluted urine that escapes from the burette contains 0·05 gramme sugar, so that one-tenth of that quantity of

undiluted urine must contain 0·05 gramme sugar. If the total quantity of urine passed in twenty-four hours be divided by the amount of undiluted urine used to reduce the solution in the capsule, and the result be multiplied by 0·05 gramme, the daily output of sugar is ascertained. The sample tested should be taken from the mixed urine of the whole twenty-four hours.

2. In *Roberts's Fermentation Test*, the sample of urine to be examined is divided into two parts, which are placed in separate dishes, side by side, in a warm place. Yeast is then added to one of the specimens only. Within twenty-four hours fermentation should be complete, and the specific gravity of each specimen is ascertained. The fermented specimen has lost much density through loss of sugar and presence of alcohol, while the other has gained a little by evaporation. The difference in degrees of density between the two represents roughly the grains of sugar per ounce.

3. In *Pavy's Ammoniated Copper Test* the suboxide is not precipitated. The observation of the complete discharge of the colour is therefore not interfered with by turbidity of the fluid.

4. *Johnson's Picric Acid Test* may be used for quantitative purposes, since the depth of the red colour is proportional to the richness in sugar of the urine examined. A standard solution of acetate of iron is used to represent the colour of urine which contains a certain proportion of sugar reduced by picric acid, caustic potash, and boiling. The amount of dilution required to bring the urine to the same colour as the standard iron solution indicates the richness in sugar.

5. The *polarimeter* can also be used in quantitative measurement, since the richer the urine is in dextrose, the further is the plane of polarised light rotated towards the right.

In diabetes the excretion of urea is increased. Moreover, certain abnormal substances, in addition to sugar, may be present in the more severe types of the disease. The most important of these are diacetic acid, acetone, and β-oxybutyric acid.

Diacetic acid in the urine is recognised by the perchloride reaction. The official solution, or the tincture of the perchloride, when added to urine containing diacetic acid, produces a deep reddish-brown or port-wine colour. A fallacy may arise with the urine of patients who are taking salicylic acid or antipyrin. In these cases the acetone test should be tried. The result is positive if diacetic acid is present, and negative if the apparent reaction with the perchloride is not due to diacetic acid.

To detect *acetone* in the urine, the nitro-prusside test is recommended by Pavy as the best for clinical purposes. To a few c.c. of the urine add a small piece of sodium nitroprusside, and then a little caustic soda. If acetone is present, a cherry-red colour is produced, which soon fades. If an excess of acetic acid is now added, a carmine-red colour appears if acetone is present.

The presence of *β-oxybutyric acid* is indicated by the different results obtained from Fehling's solution and from the polarimeter in the quantitative estimation of sugar. If no β-oxybutyric acid is present, the two methods yield practically the same result. If the acid (which is levo-rotatory) is present, it partly neutralises the rotation of the plane of polarised light which is due to the (dextro-rotatory) dextrose. In clinical work, indeed, the difference between the two results may be taken as an index of the amount of β-oxybutyric acid present.

Complications.—*Coma* is frequent and is almost invariably fatal. About half the cases of diabetes end in this way. In the most common form, the onset is rapid or almost sudden. There is abdominal pain, with languor, constipation, and perhaps vomiting. Drowsiness follows, and the breathing becomes very deep ('air-hunger'), though not much accelerated. The breath and the urine may have a sweet odour, suggestive of apples or chloroform, and due to the presence of acetone. The drowsiness deepens into coma, and death results within one or two days. In a less common form, the signs of collapse set in suddenly, especially after muscular exertion; consciousness is lost, and death takes place in a few hours. In a third and uncommon type,

there are headache, disordered gait, and increasing drowsiness, without air-hunger. Sometimes, however, the coma is uræmic, and sometimes it begins as suddenly as apoplexy. When coma sets in, the urine frequently contains albumen and numerous hyaline and granular casts. It may smell of acetone, and may contain acetone and diacetic acid as well as β-oxybutyric acid, from which both are derived. The excretion both of urine and of sugar may diminish.

Coma appears to be favoured by early life, by muscular fatigue, by obstinate constipation, by high acidity of the urine, and by a sudden change to an antidiabetic diet. It is now recognised that the characteristic coma of diabetes is due to the accumulation in the blood and tissues of β-oxybutyric acid, but this is not to be looked upon as a toxic agent which of itself poisons the tissues. It is harmful much more through its acid character, and the coma is immediately due to non-removal of carbonic acid from the tissues. This results from the *acidosis* or diminished alkalinity of the blood, and this, again, is due to the tissue disintegration which goes on in severe diabetes.

As Pavy points out,[1] excessive proteid disintegration is accompanied by excessive acid production. Thus the sulphur and phosphorus of the broken-down tissues are oxidised to form sulphuric and phosphoric acids, and these acids, together with the diacetic and β-oxybutyric acids in diabetes, neutralise much of the alkali of the blood, with the result that an acidosis is established. The probability that acidosis is the cause of diabetic coma is strongly supported by the results of experiment, and also by the fact that large doses of sodium bicarbonate have sometimes restored comatose diabetic patients to consciousness. Moreover, the coma can hardly be attributed to the direct toxic action of β-oxybutyric acid, since the acetone series of bodies is not poisonous.[2] Under normal circumstances the greater

[1] 'On the Acetone Series of Products in Connection with Diabetic Coma' (*Lancet*, 1902, ii. 64 *et seq.*).

[2] Acetone is volatile, and escapes in part by the lungs, so that it may be recognised in the breath. Traces of acetone may be present in the breath and urine of healthy persons, and the three bodies of this

proportion of the carbonic acid in the blood is conveyed from the tissues to the lungs by the alkali of the blood; the carbonic acid produced in the tissues joins the sodium carbonate of the blood to form sodium bicarbonate, and when it reaches the lungs it detaches itself again from the alkali. The carbonic acid is conveyed from the tissues by the sodium carbonate of the blood, just as the oxygen is conveyed to the tissues by the hæmoglobin. But if the excess of acid, which results in diabetes from excessive tissue disintegration, neutralises almost all the alkali, the carbonic acid will accumulate in the tissues—a condition which readily explains the air-hunger of diabetic coma.

Phthisis pulmonalis is the cause of death in about a third of the cases of diabetes. Pyrexia, cough, and other symptoms may be slight or absent, though the pulmonary lesion is advancing rapidly.

Boils and carbuncles are common. A much rarer skin complication is xanthoma. Pruritus is very common about the genitals, and may cause intense distress; it results from the deposition of glucose. Gangrene occurs in various parts, but especially in the lower limbs. Œdema of the ankles, albuminuria, neuralgia, cataract, retinitis, impotence, dental caries, pneumonia, broncho-pneumonia, and pulmonary gangrene may also be mentioned. The knee-jerks are often absent, and there may be more definite evidence of neuritis in the form of numbness, anæsthesia, pains, and paresis in the lower limbs.

Varieties.—Diabetes may be classified as *acute* and *chronic*. The *acute* form is chiefly seen in young subjects. The *chronic* form is specially common after middle life—for instance in gouty people. Another classification is into *dietetic* or *alimentary*, *nervous* or *neurotic*, and *pancreatic*. In the *dietetic* form, the inability to assimilate carbohydrates is not absolute, but appears only when more than a certain amount of such food is taken. The *neurotic* type is sup-

group may be found in the urine in other morbid conditions besides diabetes. They have the same origin, but represent different stages of oxidation. In diabetes several ounces of β-oxybutyric acid may be excreted each day.

posed to depend on disease in some part of the nervous system. In the *pancreatic* form, some evidence of pancreatic disease may be recognisable on examination of the abdomen ; or the stools may contain undigested fat. Pavy distinguishes two types, *alimentary* and *composite*. In the *alimentary* form there is a failure to assimilate carbohydrates, which thus reach the blood in the form of dextrose, and appear in the urine. In the *composite* variety there is, in addition, a retrograde tissue-metamorphosis, and this leads to the appearance in the urine, not only of dextrose, but also of bodies produced by that metamorphosis, including the acetone series of bodies. *Bronzed diabetes* is the rare variety which develops in the course of *hæmochromatosis*, a disease characterised by cirrhosis of the liver (*diabetic cirrhosis*) and pancreas, with general pigmentation ; the glycosuria ensues when the fibrosis of the pancreas leads to destruction of the islands of Langerhans.

Diagnosis.—This depends on the association of one or more of the symptoms already mentioned with the persistence of glycosuria for a considerable period of time. In practice two questions may arise : (1) whether a doubtful reaction on testing is really due to sugar ; and (2) whether the presence of slight glycosuria entitles the case to be regarded as one of diabetes. In the former case the question may be settled by the careful employment of three different tests —viz., Fehling's, the fermentation, and the phenyl-hydrazin tests—and in the latter by noting whether glycosuria is constant, and whether it immediately disappears on withdrawing carbohydrates from the diet. It should be remembered that the urine of nursing women may give a reaction with Fehling's solution, owing to the presence of lactose absorbed from the mammæ. In cases of boils, carbuncle, pruritus, cataract, and other conditions which may accompany diabetes, the urine should be examined.

Diabetic blood may be distinguished from non-diabetic blood in the following ways :

Williamson's Test.—Put 40 c.mm. of water in a narrow test-tube. Add 20 c.mm. of the blood to be tested. Then add 1 c.c. of a 1 in 6,000 watery solution of methylene blue.

Finally, add 40 c.mm. of liquor potassæ, and mix the whole thoroughly by shaking. Put the test-tube in a water-bath or large test-tube containing water ; apply heat, and keep boiling for four minutes. If the blood is diabetic, the mixture changes from a deep blue to a pale yellow colour, whereas non-diabetic blood (which should be subjected to the same treatment, at the same time, for control purposes) retains a distinct blue colour.

Bremer's Test also depends on the decolorising of aniline stains by diabetic blood. A fairly thick film is spread over half or a third of a microscope slide, which is then heated for six or ten minutes in a hot-air chamber at 125° C. The film is then stained for one and a half to two minutes in a 1 per cent. watery solution of methylene blue, Congo red, or Biebrich scarlet ; or for two to three minutes in Ehrlich-Biondi staining fluid. Methylene blue and Congo red stain non-diabetic, but not diabetic, blood. On the other hand, Biebrich scarlet stains diabetic, but not non-diabetic, blood. Ehrlich-Biondi fluid stains non-diabetic blood violet, and diabetic blood orange. With this test, also, control films of non-diabetic blood should be employed.

Prognosis.—The younger the patient the worse the outlook, as a rule. In elderly patients the disease is generally mild. The patient's carefulness as to diet, and his ability to procure suitable bread, are important elements in prognosis. If a restricted diet removes the sugar wholly or in great part, the case is promising ; whereas if the sugar persists, the emaciation continues, and the urine contains β-oxybutyric acid and its products, it is much more serious. Nevertheless, cases in which the urine yields the reactions for acetone and diacetic acid may recover, and under such circumstances these reactions disappear. Very few cases of coma recover, even under energetic treatment.

Treatment.—Great care should be taken as to suitable clothing, a healthy atmosphere, and avoidance of fatigue and worry. For the first few days the patient should be kept on an ordinary diet (except that sugar is replaced by saccharin), so that the amount of urine, sugar, etc., excreted may be ascertained. The bodily weight should also be noted at

regular intervals. Then the carbohydrates are to be gradually withdrawn.[1] It is safe for the patient to take the flesh of any kind of animal (fish, flesh, fowl) except liver, which is apt to contain sugar. Oysters are better avoided, since a large part of them consists of liver. Eggs, butter, and cream-cheese are allowable. Milk is objectionable theoretically, as containing lactose, but in practice moderate quantities (1 to 2 pints daily) may sometimes be taken with advantage. Clear soups, tea, coffee, and aerated waters are allowable if free from sugar and starchy matter. Alkaline mineral waters should be taken freely, and if the perchloride reaction is present, ½ ounce or more of sodium bicarbonate should also be taken every day. Green vegetables, tomatoes, radishes, and various pickles may be taken. Sweet fruits, beer, potatoes, turnips, and all farinaceous foods (puddings, breads, etc.) must be avoided. Special breads must be used, made from gluten, almonds, cocoanut, etc. Every fresh supply of bread should be tested for starch by iodine before it is given to the patient, as the breads on the market are sometimes quite unsuitable. After the sugar in the urine has been for some time absent or greatly reduced in amount, carbohydrates may be cautiously recommenced—say with one potato, or half a slice of bread, daily. If the patient assimilates this, the quantity may be further increased. Mild cases of the disease in wealthy patients may be sent for a time to Vichy or Carlsbad.

Of drugs, the most valuable is opium or one of its alkaloids, codeine or morphine, which may be given in increasing doses. Arsenic and sodium salicylate may be tried. Quite exceptionally benefit results from the use of a preparation of pancreas.

[1] The following dietary, adapted from Von Noorden's, is very suitable : *Breakfast :* 5 oz. ham, 1 egg, and 6 oz. tea without sugar or milk. *Lunch :* 6 oz. meat ; 2 oz. cucumber with 1 dr. vinegar, 2 dr. olive oil, and pepper and salt to taste ; 13 oz. soda-water ; and 6 oz. coffee without sugar or milk. *Dinner :* 10 oz. clear soup ; 8 oz. meat basted with 2 oz. butter ; 2 oz. green salad, with 2 dr. vinegar and 5 dr. olive oil, or 1 oz. well-cooked green vegetable ; 3 sardines with oil ; 13 oz. soda-water. *Supper :* 2 eggs, raw or cooked ; 13 oz. soda-water.

For thirst, lemon-juice or a bitter vegetable infusion may be given. For itching of the genitals, the parts should be sponged and dried after micturition ; and if this is insufficient, they may be bathed with a lotion containing sulphurous acid, or anointed with ointment of conium.

It is important to avoid constipation. If coma threatens, a carbohydrate diet should be reverted to, and large quantities of alkali should be given till the urine becomes neutral or faintly alkaline. When coma sets in, an alkali should be injected into the veins ; Naunyn recommends 9 or 10 drachms of sodium carbonate[1] dissolved in $1\frac{3}{4}$ pints of water (35 to 40 grammes in 1 litre). Next to intravenous infusion, the best treatment is the daily administration of alkalies by the mouth (*e.g.*, from $2\frac{1}{2}$ to 4 ounces of sodium carbonate).

N.B.—Experts differ in the stringency with which they select a dietary for diabetic patients. The following lists, modified from a table of Pavy's, may be useful :

Allowed.—Butcher's meat of all kinds, except liver ; ham, bacon, and other preserved meats ; poultry and game ; fish of all kinds, fresh and cured ; claws of crabs and lobsters ; animal soups, beef-tea, and broth (not thickened) ; eggs ; cream, butter, cheese, and cream-cheese ; almond, bran and gluten substitutes for bread ; greens, spinach, turnip-tops, water-cress, mushrooms, mustard-and-cress, cucumber, lettuce, endive, spring onions, radishes, and celery.

Allowed in Moderate Quantity (*the Vegetables should be boiled in much Water.*)—Turnips, French beans, Brussels sprouts, cabbage, cauliflower, broccoli, seakale, asparagus, and vegetable-marrow ; pickles, olives, vinegar, and oil ; jelly (flavoured, but not sweetened), savoury jelly, blanc-mange (made with cream and not with milk), and custard (made without sugar) ; nuts (except chestnuts).

[1] Sodium bicarbonate has been generally used, and is still recommended. But it is obvious, when the nature of acidosis is considered (see pp. 246, 247), that if the carbonate is tolerated by the body, it ought to be much more efficient than the bicarbonate as a neutraliser of the acid in the blood. The strong solution of the carbonate recommended by Naunyn must not be injected subcutaneously, as it almost always causes gangrene ; but it seems to be well borne, and to act well when introduced into the veins.

Beverages Allowed.—Tea, coffee, and cocoa from nibs ; dry sherry, claret, hock, dry Sauterne, Chablis, and Burgundy ; brandy and spirits (unsweetened) ; soda-water.

Beverage Allowed in Moderate Quantity.—Burton bitter ale.

Forbidden.—Sugar in any form ; wheaten bread and ordinary biscuits ; rice, arrowroot, cornflour, sago, tapioca, macaroni, and vermicelli ; potatoes, carrots, parsnips, beetroot, peas, and Spanish onions ; pastry and puddings ; all fruits, whether fresh or preserved ; liver ; oysters, cockles, mussels, and the pudding of crabs and lobsters.

Beverages Forbidden.—Milk (except sparingly) ; sweet ales, porter, stout, and cider ; sweet and sparkling wines ; port wine (except sparingly) ; rum and sweetened gin ; liqueurs.

9. DIABETES INSIPIDUS.

Definition.—A disease characterised by the persistent passage of large quantities of urine free from sugar and albumen.

Etiology.—The disease may occur at any age, but is met with chiefly in the first half of life. It may be inherited through several generations. Males suffer more than females. Among the recognised causes are injuries of the head or other parts, emotion, and cerebral disease. Alcoholism, acute disease (*e.g.*, influenza), and large draughts of cold fluids are also causes, but it is possible that a large draught is really an expression of the first symptom, viz., thirst. Persistent polyuria is met with in association with organic brain disease, and in a good many cases the latter is of syphilitic nature (*e.g.*, gumma or meningitis).

Cases where there is no evidence of underlying organic disease are distinguished as *primary* or *idiopathic ;* while those in which there is structural disease in the nervous system or elsewhere are spoken of as *secondary* or *symptomatic.*

Morbid Anatomy.—There is no characteristic change. In the secondary cases, intracranial disease has often been found. The bladder and kidneys are sometimes hypertrophied as a consequence of the excessive work they have to do. Occasionally the ureters are dilated and the kidneys sacculated.

Pathology.—The disease is supposed to be a paralysis of

the vasomotor nerves of the kidneys, resulting from functional or structural changes in the region of the fourth ventricle, or from disease involving the splanchnic nerves. Bernard found that polyuria resulted from a puncture in the floor of the fourth ventricle above the level of the diabetic (glycosuric) puncture.

Symptoms.—The onset is gradual or sudden. The principal symptoms are great increase in the quantity of urine, and, as a consequence, great thirst. The quantity has been known to exceed 50 pints daily, and 15 to 40 pints are not uncommon. In secondary cases, however, the excretion may be 5 to 10 pints. The specific gravity is from 1001 to 1007, and the appearance suggests pure water. The daily excretion of urinary solids may be practically that of health. In many cases the general health is unaffected, but in others there are symptoms resembling those of mild diabetes mellitus—dry skin, abdominal pains, excessive appetite, impotence, and general weakness. The duration varies from a few weeks to more than half a century.

Diagnosis.—*Hysterical polyuria* is transient, and is recognisable by the associated symptoms.

In *chronic interstitial nephritis* there are hyaline casts, and at times, if not constantly, albumen in the urine; moreover, cardiovascular and sometimes retinal changes are present.

Amyloid disease of the kidneys is also recognisable by the albuminuria and concomitant symptoms.

In *diabetes mellitus* the urine contains sugar and has a high specific gravity

Prognosis.—In secondary cases, this depends upon the lesion—*e.g.*, injury to or tumour of the brain. In primary or idiopathic cases, cure may take place even after many years' duration. Death is almost always due to intercurrent disease.

Treatment.—This is seldom satisfactory in primary cases. Valerian in some form, or valerianate of zinc may be tried in increasing doses. Ergot, antipyrin, strychnine, arsenic, and other tonics, as well as the bromides, have been recommended. Opiates appear not to be beneficial, but rather the reverse. Galvanism may be employed, one pole being

placed on the loin, first of one side and then of the other, and the opposite pole in the hypochondrium or at the occiput ; or the negative pole may be applied to the posterior wall of the naso-pharynx, while the positive pole is applied to the nape of the neck. A change of air may prove useful.

10. OBESITY (Adiposity. Corpulence).

The etiology of this condition is not fully understood. The tendency to it is sometimes inherited, and in those who are predisposed its development is favoured by over-eating, want of exercise, and alcoholism. But it is by no means always due to overeating. Chlorotic girls are often stout. Women after the menopause and men after middle life often become corpulent.

Gout is probably a cause. The effects of corpulence vary much in different persons. Sometimes there is great increase of the epipericardial fat, and this may extend between the muscle-fibres and hamper the cardiac action. The fat on the walls of the chest and abdomen, and in the omentum, embarrasses the action of the respiratory muscles. Bronchitis and other acute diseases are not well borne. Very obese young subjects may show a strong tendency to sleep.

Treatment.—Excesses in eating and drinking must be stopped, and, wherever practicable, regular physical exercise must be taken. Various dietaries have been introduced, most of them reducing the total amount of food taken, but especially reducing the carbohydrate element. Thus, Banting's method, formerly popular in England, excluded farinaceous foods, sugar, and fats, and restricted the liquids. The diet consisted of about 1 pound of animal food, with several ounces of fruits and vegetables, and only 2 ounces of bread. The total fluid was 35 ounces. Ebstein gives a dietary which, while spare in quantity, is rich in fat, on the ground that fat gives rise to a feeling of satisfaction and diminishes thirst, thus lessening the desire for both solids and liquids ; sugar, sweets, and potatoes are forbidden. Oertel allows more fat than Banting, and much more proteid and carbohydrate than Ebstein. He restricts

the liquids, and gives the solids and liquids at different times. Moreover, he tries to strengthen the cardiac muscle by regulated exercise in walking or hill-climbing. Schweninger also restricts the liquids, gives a lean and spare diet, and employs massage and gymnastics, so that his reduction cure is a fairly severe one. There is good reason to believe that restriction of fluids ('thirst cure') has no direct influence on the destruction of fat in the body, although the deprivation of liquids at meal-times doubtless causes many people to eat less solid food than they would otherwise take. Weir Mitchell recommends rest, a diet of skimmed milk, and massage. In the Salisbury method, the diet is restricted for a time to rumpsteak, codfish, and hot water. Whichever method is adopted, it should be employed with caution, and in some people no treatment may be desirable. In some cases thyroid extract (5 grains thrice daily) is beneficial, but its effects should be carefully watched by the physician.

In the milder degrees of obesity, a reduction cure should not be undertaken without due consideration, as the last state may actually be worse than the first. The loss of support to the abdominal viscera may lead to constipation, displacement of the kidneys or of the uterus, gastroptosis, and hernia. Hepatic colic is a frequent sequel, according to Von Noorden, and is explained by the biliary organs being no longer protected by the layer of fatty tissue from the pressure of corsets and waistbands.

11. ADIPOSIS DOLOROSA (Dercum's Disease).[1]

This is a rare affection, met with principally in middle-aged women. There is usually a history of alcoholism or of syphilis. There are multiple, more or less symmetrical, fatty tumours under the skin of the arms and other parts, but not of the face, hands, or feet. Neuralgic pains accompany or precede the swellings, and the latter may be tender. Mental symptoms may be present. The thyroid body has occasionally been diseased, and improvement has been observed under treatment by thyroid extract.

[1] Described by Dercum in 1889.

SECTION III

DISEASES OF THE CIRCULATORY SYSTEM

1. ANATOMY.

The following anatomical facts should be borne in mind :

Under normal circumstances the upper limit of the heart corresponds to the level of the second costal cartilages. The lower limit extends from the seventh right costal cartilage at its sternal end to the apex. The right border extends from the second to the seventh right chondro - sternal junction, and is convex to the right, so that it reaches as far as $1\frac{1}{2}$ inches to the right of the mid-line. The left border passes from the second left cartilage to the apex with a convexity upwards. The apex is in the fifth left intercostal space about $3\frac{1}{2}$ inches from the mid-line. It is usually felt beating below and a little internal to the nipple. The area indicated above corresponds theoretically to the *deep cardiac dulness*.

The *superficial cardiac dulness* corresponds theoretically to the part of the heart which is not covered by lung. The right border of this area is at the middle line, but as the sternum is an excellent sounding-board, it is usually recognised on percussion as at the left edge of the sternum. The dulness merges below into the hepatic dulness. The upper border is somewhere from the third to the fourth left costal cartilage. The outer border is slightly internal to the nipple line.

The pulmonary artery corresponds in part to the second left space, and extends up to the second left cartilage.

Sounds generated at the pulmonary valve are examined at the second left space (or cartilage—pulmonic cartilage). The aortic cartilage (second right) corresponds to the

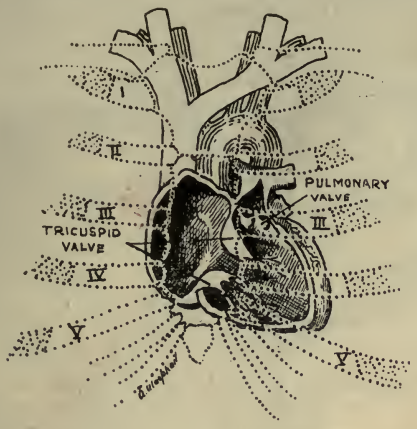

FIG. 13.—SECTION OF THE HEART, SHOWING THE RIGHT AURICLE AND RIGHT VENTRICLE, WITH THE TRICUSPID AND PULMONARY VALVES, IN THEIR RELATION TO THE STERNUM, COSTAL CARTILAGES, AND GREAT VESSELS.

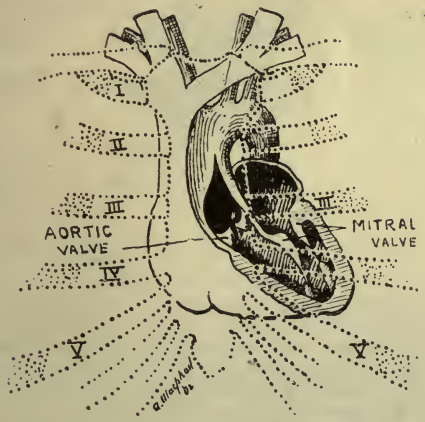

FIG. 14.—SECTION OF THE HEART, SHOWING THE LEFT AURICLE AND LEFT VENTRICLE, WITH THE MITRAL AND AORTIC VALVES, IN THEIR RELATION TO THE STERNUM, COSTAL CARTILAGES, AND GREAT VESSELS.

junction of the ascending and transverse portions of the aortic arch. The transverse part of the aortic arch corresponds to the lower half of the manubrium. The left

innominate vein passes to the right behind the upper half of the manubrium, and in front of the three large branches of the aortic arch.

The superior vena cava passes from the first to the third

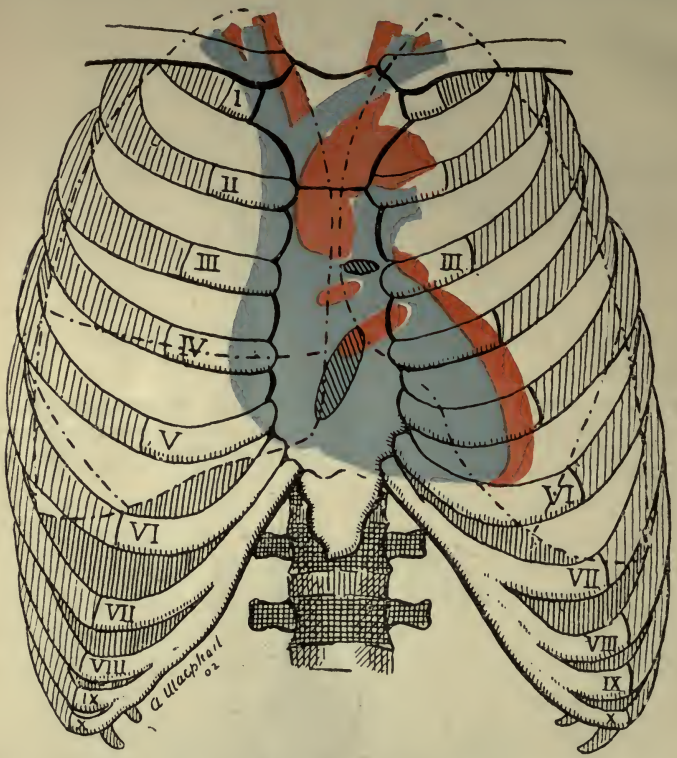

FIG. 15.—RELATIONS OF THE HEART AND GREAT VESSELS TO THE LUNGS AND THORACIC WALL.

The figure shows the relations of the heart and great vessels to the anterior wall of the chest; the outline of the sternum, costal cartilages, and ribs; and the anterior margins of the lungs with the interlobar fissures.

The aortic arch is seen behind the lower half, and the left innominate vein behind the upper half of the manubrium sterni.

Of the shaded oval areas, the larger represents the tricuspid and the smaller the pulmonic valve.

Of the red oval areas, the larger represents the mitral and the smaller the aortic valve.

The blue colour corresponds to venous and the red to arterial blood.

right costal cartilage, perforating the fibrous pericardium half-way down.

A line from the third left to the fifth right costal cartilage marks off the right auricle from the right ventricle.

2. SYMPTOMATOLOGY.

Some of the symptoms of cardiac disease are exactly the same as those which appear in a healthy person after violent exercise which overtaxes the reserve strength of the heart. Others represent a more severe strain on the heart, such as is never seen in a perfectly healthy individual.

Dyspnœa, or difficulty in breathing, is common, and is often the first symptom of heart disease. It is induced or aggravated by exertion. Sometimes it is so severe that the patient cannot lie down (*orthopnœa*). The comparative relief obtained by sitting up is in part accounted for by the fact that the descent of the diaphragm allows the abdominal viscera simply to move forwards in inspiration, whereas when the patient lies on his back these viscera have to be partly raised against gravity.

Occasionally the dyspnœa is paroxysmal. Sudden and severe attacks of dyspnœa set in without obvious cause (*cardiac asthma*).

Cheyne-Stokes respiration (*periodic* or *tidal breathing*, *rhythmical dyspnœa*) is rhythmical in its character. Breathing is arrested altogether for a time, and then recommences with shallow inspirations. These become deeper and deeper, until at the acme they are extremely strong, noisy, and distressing. After a short time they gradually become shallower, and at length cease altogether for a time. The details of the cycle vary greatly even in a given case. In a well-marked example the cycle may extend over three-quarters of a minute, of which twelve seconds may be occupied by the pause, or interval of apnœa. While breathing is actually going on, it may be slow or rapid—*e.g.*, at a rate of 20 per minute, or at a rate of over 80 per minute. The period of dyspnœa, with its rise and decline, is usually longer than the period of apnœa.

Cheyne-Stokes respiration may be as striking when the patient is awake as when he is asleep. The slighter degrees do not prevent sleep, but in more marked cases the patient, if drowsy or sleeping, is wakened at or before the acme of each respiratory cycle by the violence of the dyspnœa. In

a patient who is wide awake and fully conscious, the more severe forms of this respiratory disturbance give rise to an aspect and movements indicative of intense distress, though there is no actual pain. The slighter degrees seem to pass unnoticed by the patient, and even in severe types the complete and prolonged apnœa of the interval causes no discomfort.

Full mental power may be preserved in spite of Cheyne-Stokes breathing, although the intelligence is often dulled by the cause of the respiratory disorder. The pupil may contract in the stage of apnœa and dilate in that of dyspnœa. It has been noticed to change its size with each powerful respiration. The pulse may vary with the different periods of the cycle, or may remain perfectly uniform.

Cheyne-Stokes breathing is met with under many different circumstances, including diseases of the brain and membranes, heart and great vessels, lungs and kidneys ; also in connection with acute fevers, sunstroke, morphine poisoning, senility, and apparently complete health. In diseases of the heart, brain, and kidneys, and in aneurysm, it is not a favourable sign, but the patient may survive for a considerable time, and may temporarily recover from this rhythmical dyspnœa. In a patient rescued from morphine poisoning, permanent recovery may of course be witnessed.

Periodic respiration is sometimes classified into two types, the one being *Cheyne-Stokes breathing*, which has just been described, and the other being known as *grouped breathing*. In the latter variety the respirations are slow and of equal range, and occur in groups of two or three. The significance of this form is not so grave as that of the Cheyne-Stokes type.

In spite of all the theories that have been suggested, Cheyne-Stokes respiration is still unexplained. It is a curious fact that whilst poisoning by morphine will induce Cheyne-Stokes breathing, a dose of that drug will occasionally remove the symptom for a time, even when it is very marked.

The dyspnœa of cardiac disease is largely due to defective oxygenation of the respiratory centre in the medulla. The

various valvular lesions; weakness of the cardiac muscle; congestive, catarrhal and dropsical conditions of the lungs and bronchi; and sometimes obstruction of branches of the pulmonary artery, interfere mechanically with the aeration of the blood, and thus stimulate the respiratory process.

In aneurysm, dyspnœa may be due to pressure on the trachea, bronchi, or nerves. It may be paroxysmal.

Palpitation means rapid and violent action of the heart of which the patient is conscious. There is generally some uneasiness in the cardiac region, and there may be pain, faintness, and alarm. Many of the beats of the heart may fail to reach the wrist, owing to the ventricle not taking time, either to fill itself completely from the auricle, or to empty itself completely into the aorta. Common as it is in heart disease, palpitation is still more common in less serious conditions, such as dyspepsia, emotional excitement, etc. It is one of the type-symptoms of Graves' disease. The palpitation of heart disease is specially apt to be induced by exertion.

Disturbances of rhythm of the cardiac action are common in advanced heart disease, and are often described by patients as a 'fluttering at the heart.' When the heart's action is regular for a number of beats in succession, and only one beat is missed from time to time, it is described as *intermittent*. Disturbance of rhythm is one of the common neuroses of the heart.

Pain in the chest is as often absent as present in well-marked heart disease, and when it is present it is not, as a rule, referred exactly to the cardiac region. There may, however, be intense pain in pericarditis, and one of the neuroses of the heart, angina pectoris, has cardiac pain as its principal element. Pain about the heart is very often complained of by people whose hearts are perfectly healthy. Such pains are often due to dyspepsia, neuralgia, pleurisy, and pleurodynia. Sudden pain in the chest in heart disease may result from pulmonary embolism.

Cough is very common in heart disease, and is often the result of secondary changes in the respiratory system, such as congestion and catarrh of the bronchial passages, con-

gestion and œdema of the lungs, etc. Sometimes, however, it appears to be purely reflex. In aneurysm of the aorta, cough may be present, and may have a peculiar quality, owing to paralysis of an adductor muscle of the larynx.

Hæmoptysis is common in advanced heart disease, and is usually due to pulmonary congestion, but may be due to infarction. Considerable bleeding is specially associated with mitral stenosis. Aortic aneurysm is an important cause of hæmoptysis.

Abnormal pulsations are often seen in heart disease and in aneurysm. When the right ventricle is much dilated there may be extensive heaving of the chest-wall in front. The carotids and other arteries beat excessively in the aortic regurgitation of young subjects, and in the same condition pulsation may extend into the capillaries. In tricuspid regurgitation there may be pulsation in the veins of the neck, and occasionally in the liver itself owing to pulsation of the hepatic veins.

Cyanosis is common as a result of deficient oxygenation. Its extreme form is observed in connection with congenital heart disease. Lividity is often combined with pallor.

Pallor is specially striking in the severe heart disease of children, and in the aortic regurgitation of adolescents and young adults.

Clubbing of the ends of the fingers is sometimes very striking in congenital heart disease. Examination of such a finger may show an increase of fibrous tissue, especially around the vessels. The condition is apparently a result of passive hyperæmia. It is occasionally seen in heart disease in the adult, particularly in mitral stenosis.

Dropsy is common when compensation fails. Cardiac dropsy begins in the most dependent parts, but in advanced cases is often widespread, involving the serous sacs as well as the cutaneous, subcutaneous, and other tissues.

Gastric catarrh, *hæmorrhoids*, and *enlargement of the liver*, sometimes with slight jaundice, result from passive hyperæmia of the digestive system.

Among *nervous disturbances* are insomnia, delirium, and muscular weakness.

Syncope is specially liable to occur in aortic regurgitation.

Hemiplegia may result from embolism, which is specially common in mitral stenosis.

Owing to passive hyperæmia of the kidneys, the urine is concentrated, and *albuminuria* is common.

3. THE PULSE.

In examining the radial pulse by the finger, it is important to note (1) the frequency; (2) the regularity (*a*) of force and (*b*) of rhythm; and (3) the quality, viz., (*a*) the size, (*b*) the tension, and (*c*) the mode of expansion.

Disturbances of frequency and regularity are met with as cardiac neuroses (described later on); but rapid action and irregularity of force and rhythm are frequently observed when the cardiac action is failing.

The size of the pulse varies much. When the heart is failing, the pulse is generally small and rapid.

A pulse of *high tension* is hard, and difficult to obliterate by pressure. It means that the arterial outflow is difficult.[1] It is met with in kidney disease, gout, lead-poisoning, constipation and early anæmia, and is due probably to abnormal material in the blood increasing the resistance in the capillaries. It is found also when the arterioles are constricted by the application of external cold; in the cold stage of fevers; and in connection with hypertrophy of the left ventricle, owing to the increased *vis a tergo*. A small and hard (wiry or thready) pulse is met with in peritonitis.

A pulse of *low tension* is soft and easily obliterated. If the tension is very low, *dicrotism*, or doubling of the beat, may be detected by the finger at the wrist. In low tension, the outflow from the arterioles into the veins is free. This pulse is met with when the arterioles are relaxed—for instance, by warmth or by use of amyl nitrite. It is also seen in the later stages of anæmia and fevers, and in other conditions characterised by enfeeblement of the cardiac muscle.

[1] An artery which cannot be obliterated owing to the high pressure of blood within it must be distinguished from an artery which is rigid owing to disease of its wall. The two conditions may coexist.

The pulse expands slowly in aortic stenosis and in other conditions where there is abnormal resistance to the work of the ventricle, and where at the same time the ventricle is competent. The expansion is sudden in the pulse that is characteristic of aortic regurgitation (*Corrigan's pulse ; water - hammer*, *splashing*, or *collapsing pulse ; pulse of*

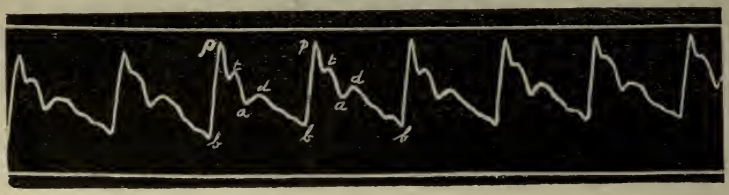

FIG. 16.—PULSE OF LARGE VOLUME.

b, b, b, respiratory or base line ; *p*, primary or percussion wave; *t*, secondary, tidal, or predicrotic wave ; *d*, dicrotic wave ; *a*, dicrotic or aortic notch.

unfilled arteries) ; this pulse is suddenly filled, but collapses between the beats.

In addition to more markedly irregular pulses, there are the *intermittent pulse*, which simply misses a beat now and then ; the *pulsus bigeminus* (Fig. 24, p. 268), in which the beats come in couples ; and the *pulsus trigeminus*, with the beats in groups of three. In the *pulsus paradoxus* the pulse

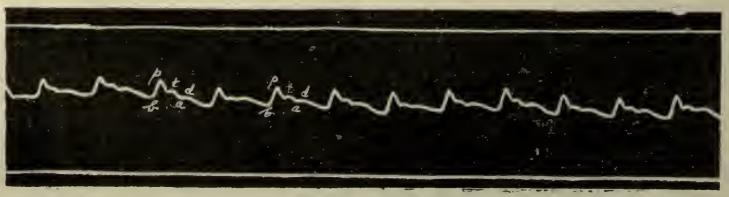

FIG. 17.—PULSE OF SMALL VOLUME.

b, b, b, respiratory or base line ; *p*, primary or percussion wave ; *t*, secondary, tidal, or predicrotic wave ; *d*, dicrotic wave ; *a*, dicrotic or aortic notch.

is almost or completely lost in inspiration, to reappear with expiration. It may sometimes be due, as was once supposed, to mediastino-pericarditis, with the formation of bands which are drawn tight over the great vessels in inspiration ; but it has been observed in various other diseased conditions, and even in health.

Aortic aneurysm, as will be explained hereafter, is frequently accompanied by differences between the two radial pulses.

The SPHYGMOGRAPH is of value in enabling us to study the pulse-wave in detail. Under normal conditions, this wave (Figs. 16 and 17) is made up as follows: Starting

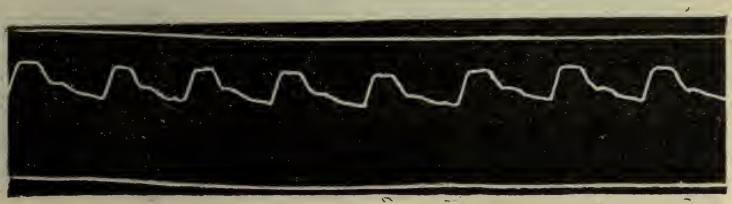

FIG. 18.—ANACROTIC PULSE TRACING, SHOWING INCREASED RESISTANCE TO ARTERIAL OUTFLOW. (Chas. McG., æt. 45, V.S., V.D. aortic murmurs.)

from the *respiratory* or *base line*, there is first the *primary* or *percussion wave*, and this is soon followed by the *secondary*, *tidal* or *predicrotic wave*, which represents the actual blood-tension within the vessel. The primary and secondary waves ought in theory to be one and the same; but the momentum given to the lever of the sphygmograph by the suddenness of the pulse carries the needle too far. The third wave is the *dicrotic wave*, which is due to the recoil of the aorta after being distended by the blood thrown into

FIG. 19.—HIGH-TENSION PULSE. (Geo. B., æt. 38, acute nephritis.)

it by the ventricle. After the tidal, and before the dicrotic wave, is the *dicrotic* or *aortic notch*, which corresponds in time to the closure of the aortic valve. Smaller waves of no special importance may be recognised after the dicrotic wave, as the tracing gradually falls to the base line. The *respiratory* or *base line* varies in height with deep inspira-

tions and expirations, and represents the residual tension in the vessel, namely, the tension between the beats (Fig. 23). The tracing from the commencement of the percussion wave to the summit of the tidal wave corresponds to the *systole of the ventricle*, and the *first sound* of the heart. From the summit of the tidal wave till the tracing reaches the base

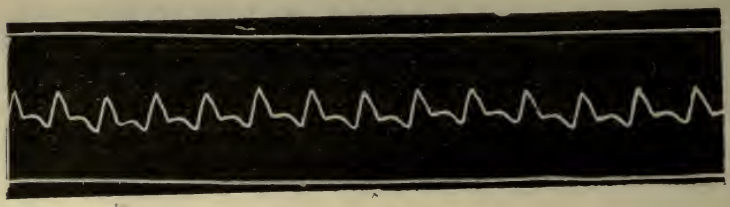

Fig. 20.—Dicrotic Pulse. (J. P., æt. 35, pneumonia, 6th day, pulse 108.)

line corresponds to the *diastole of the ventricle*. The *aortic* or *dicrotic notch* corresponds to the *second sound*.

Sphygmograms are divisible in the first place into *katacrotic* and *anacrotic*. In the former group the percussion wave shows an unbroken line of ascent, and has a pointed top, whereas in the latter the percussion wave is broken in its ascent, and the top is rounded or flat. The pulse-tracings of normal tension (Fig. 16) and of low tension (Fig. 20) are katacrotic, while anacrotic tracings (Fig. 18)

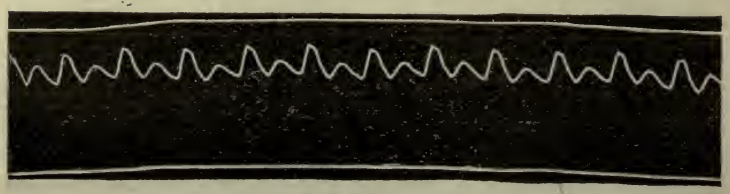

Fig. 21.—Full Dicrotism. (Janet S., ? enteric.)

are met with in certain cases where the left ventricle meets with increased resistance to its work, as in aortic stenosis and in chronic interstitial nephritis. In acute nephritis (Fig. 19) the tension is often high, as shown by the prominent tidal wave ; and as the tidal wave approaches or surpasses the primary wave in height, the tracing becomes transformed from the katacrotic to the anacrotic type. If, under con-

ditions of high tension, the left ventricle fails, the tension will then fall, and the sphygmogram will again be transformed.

In the pulse of *high tension* (which may be either large or small), the tidal wave is high, and the dicrotic wave is

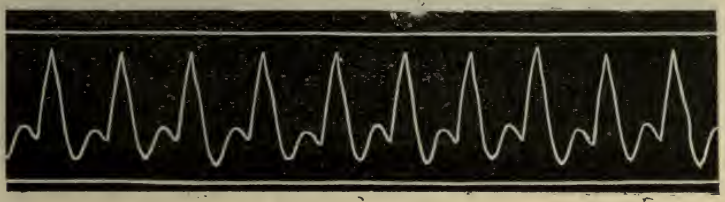

FIG. 22.—HYPERDICROTISM. (Jas. M., æt. 20, pneumonia, 6th day, pulse 108.)

inconspicuous, though it may be high up in the diastolic line (Fig. 19).

In the pulse of *low tension* (Figs. 20 to 23) the dicrotic wave is unduly prominent, there is little or no tidal wave, and the aortic notch is low. *Dicrotism*, then, means low tension. In *full dicrotism* (Fig. 21) the tension is so low that the aortic notch is at the base line. In a case where the tension is so low as this, when the heart is acting rapidly, a new

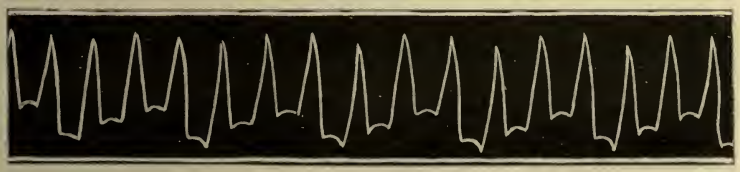

FIG. 23.—SPHYGMOGRAM SHOWING LOW-TENSION PULSE. (Mrs. K., pneumonia.)

The influence of respiration on the base line is very marked. The pulse-respiration ratio is 3 to 1, instead of the normal 4 to 1. The waves are arranged in groups of three, and the different waves in a group illustrate in a striking way different degrees of dicrotism.

primary wave may commence before the dicrotic wave has subsided to the level of the dicrotic notch. The dicrotic notch is therefore lower than the commencement of the percussion wave. Such a condition is termed *hyperdicrotism* (Fig. 22). In a still further development of this

condition, the percussion wave begins at the summit of the dicrotic wave, before the latter has even begun to subside, so that the primary and dicrotic waves are blended into one. Such a pulse is described as *monocrotic*. Low-

Fig. 24.—Pulsus Bigeminus, or Pulse with Coupled Beats.

tension pulses are observed specially in the late stages of fevers—*e.g.*, pneumonia.

The pulses of valvular disease of the heart and of aneurysm will be described in connection with the diseases with which they are associated.

4. PHYSICAL EXAMINATION.

Inspection.

In disease as well as in health, it is often possible to see pulsation in a limited portion of the cardiac area corresponding to the region of the apex. This normal apex beat is generally, though not by all,[1] understood to be due to the left ventricle. It is described as punctuate, on account of its limited extent; but if the heart is dilated, pulsation may be widely diffused over the front of the chest. The pulsation is then often due to the right ventricle.

The normal impulse corresponds in time with the systole of the ventricle. Its position is usually in the fifth intercostal space, a little distance internal to the nipple line. Its situation varies, however, even in health, and in children may be both higher and further to the left than in adults. In disease, great changes are met with. Enlargement of the left ventricle causes the apex beat to be shifted downwards and to the left. Enlargement of the right ventricle causes an increase in the area of

[1] Thus Gee says *right* ventricle; 'Auscultation and Percussion,' fourth edition (1893), p. 39.

pulsation towards the right, but it may also cause displacement of the apex beat to the left. Pulmonary emphysema may cause the cardiac impulse to disappear altogether. Pleural effusion or pneumothorax may give rise to great displacement of the heart as a whole, either towards the right or towards the left.

Systolic pulsation which is sometimes seen in the inner portion of the second left space is referable to the pulmonary artery.

Pulsation to the right of the sternum may be due to a dilated right auricle, or to an aneurysm.

Systolic recession of intercostal spaces may result from atmospheric pressure, especially if the heart is much hypertrophied. If the costal cartilages and lower end of the sternum are drawn in at the systole, pericardial adhesion is probable.

Great enlargement of the heart and pericardial effusion may lead to a local bulging of the chest-wall.

Other points to be attended to are : distension of superficial veins, pulsation of veins, bulging or pulsation in the possible situation of an aortic aneurysm, and excessive pulsation of arteries.

Palpation.

Palpation is used in counting the ribs, an art which many students appear to have great difficulty in acquiring. Palpation enables us to fix the position of the apex beat more accurately than we can do by inspection. It gives us information as to the manner in which an enlarged ventricle is doing its excessive task. It enables us to study the pulsation of aneurysms, and the enfeeblement of the cardiac impulse in pericardial effusion and in pulmonary emphysema. It enables us to fix the rhythm of various phenomena—*e.g.*, murmurs ; the instant at which the apex transmits its impulse to the finger indicates the ventricular systole. A slight diastolic shock due to closure of the pulmonic valves is occasionally to be felt over the pulmonary artery. A diastolic shock may also be due to aortic aneurysm, adherent pericardium, or cardiac hypertrophy.

When the right auricle is much enlarged, it may give rise to a presystolic impulse.

Palpation also reveals valvular *thrills*. These are vibrations generated at the valve orifices, and are similar in origin, nature and significance to the valvular murmurs which are detected by auscultation. The sensation has been compared to that felt by a hand placed on the back of a purring cat. The most common thrill is presystolic in rhythm, is situated at the apex region, and is due to mitral stenosis. Aortic aneurysm may also give rise to thrill. Pericardial friction-fremitus is the sense of rubbing felt when the hand is placed over the heart in some cases of pericarditis.

Percussion.[1]

The chest may be struck directly by the fingers ; or the finger or some rigid body (the *pleximeter*) may be closely applied over the part to be examined and then struck by the finger or other striking body (the *plessor*). The former method is *immediate*, the latter is *mediate* or *intermediate* percussion. In examining the heart, mediate percussion is the method almost always employed, and as a rule a finger of one hand is used as the plessor, and a finger of the other hand as the pleximeter. Percussion is carried out in the direction towards the heart, starting from the air-filled viscera of the thorax and abdomen.

As an index of the actual size of the heart, the *deep* or *relative cardiac dulness* is theoretically much more valuable than the *superficial* or *absolute dulness*, but in practice it is far otherwise ; and as a rule the latter only need be carefully studied. For the delimitation of the superficial dulness, light percussion must be used.

In cases where the apex beat cannot be seen or felt, the left border and the left end of the lower border may be ascertainable by percussion. Delimitation of the cardiac dulness is of great importance in enabling us to recognise enlargement of the different chambers of the heart, displace-

[1] Reference may be made here to the remarks on percussion in the introduction to Diseases of the Respiratory System (Section V.).

ment of the heart, and pericardial effusion. The area is increased in enlargement of the heart and in pericardial effusion, and is diminished in pulmonary emphysema.

Percussion may reveal abnormal dulness in a certain part of the chest in consequence of the presence of an aortic aneurysm.

Auscultation.

Auscultation may be *immediate* or *mediate* (*intermediate*). The latter form, in which a stethoscope is employed, is most generally useful, but sometimes in the case of timid children the ear may be applied to the chest directly. For accurate work, however, and in the case of the heart even more than in that of the lungs, a stethoscope is essential.

Auscultation is employed in the examination of the sounds of the heart, which may undergo different kinds of modification ; and in the investigation of two classes of adventitious sounds, viz., endocardial murmurs and pericardial friction sound. Friction sound is described in connection with pericarditis.

The *second sound* of the heart is due to the sudden tension in the aortic and pulmonic valves, and in the neighbouring portions of the aorta and pulmonary artery, at the instant when these valves close. Accordingly if the aorta is dilated the pitch of the aortic second sound is lowered.

The second sound is much shorter than the first, and to the ear the character of tension is manifest enough, but there is no element of duration or prolongation.

The *first sound* is attributable chiefly to two factors. It is partly a muscular sound, due to the contraction of the ventricles. This sound is of some duration, and explains the element of prolongation in the first sound. Another factor in the production of the first sound is the tension induced in the auriculo-ventricular valves especially, but also in the walls of the ventricles, when the chambers contract upon their contents. This gives rise to the tension element in the first sound, and if the heart is dilated and failing, this tension element may alone be audible, so that the first sound is then like a normal second sound.

The four valves of the heart are clustered together pretty closely, chiefly under cover of that portion of the sternum which extends from the second to the fourth intercostal spaces. Indeed, it has been estimated that they all lie under a square half-inch of surface. The consequence is that auscultation over the exact site of any particular valve would (except in the case of the pulmonic) yield a sound produced in part at the closely adjacent valves. For this and other reasons, advantage is taken of the conduction of sounds produced at the individual valves to recognised areas on the surface.

Sounds produced at the *mitral* valve are conducted by the left ventricle to the apex, which of course comes closely in contact with the chest-wall when the ventricle contracts.

Sounds produced at the *tricuspid* valve are heard at the left border of the sternum, at about the level of the apex, or near the base of the xiphoid cartilage.

Sounds produced at the *aortic* valve are studied where the aorta comes to the surface, viz., at the second right costal cartilage (aortic cartilage).

Sounds produced at the *pulmonic* valve are (alone of the valve sounds) examined over the immediate site of production, viz., at the inner end of the second left intercostal space.[1]

Changes in the characters of the cardiac sounds are common, and are often of importance in diagnosis and prognosis. In severe exhaustion, as in acute fevers, the first sound at the apex may be greatly enfeebled or even lost. The sounds generally may be very faint owing to emphysematous lungs overlapping the heart. A quiet, deliberate first sound is heard in hypertrophy, and a short, sharp first sound suggests the failure of a dilated ventricle. In advanced mitral stenosis, a short, sharp first sound may be the only sound of cardiac origin audible at the apex.

Accentuation of the Second Sound.—The pulmonic second sound is accentuated in mitral disease, whether obstructive or regurgitant. This is due to the increased tension in the

[1] In some instances the pulmonic sounds may be better heard in the *third* left space.

pulmonary artery which results from the lesion in front, and from hypertrophy of the right ventricle behind. If the ventricle should at length fail, the second sound will lose in intensity.

The aortic second sound is accentuated when the tension in the arterial system is increased. The pitch of the aortic second sound is lowered when the root of the aorta is dilated.

Reduplication of the Heart's Sounds.—Reduplication of the first sound at the apex is due to the two ventricles not contracting simultaneously. This sometimes occurs when the left ventricle is beginning to fail under the prolonged abnormal strain of high arterial tension or aortic stenosis. There is some uncertainty as to whether the embarrassed ventricle contracts too soon or too late.

Reduplication of the second sound at the base means non-simultaneous closure of the aortic and pulmonic valves. When persistent, it is specially suggestive of mitral stenosis, in which condition the increased pressure in the pulmonary artery causes the pulmonic valve to close before the aortic.

Cardiac murmurs are of incalculable value in enabling us to recognise the existence of heart disease, and in enabling us to decide which valve is at fault. When a fluid passes from a tube of given calibre into one of larger calibre, or through an orifice into a wider space, that fluid is thrown into eddies. These eddies constitute a 'fluid vein,' and in the case of blood in the heart their vibrations are often recognisable by the ear as a murmur, or by the hand as a thrill. If a valve orifice is narrowed by disease, a fluid vein is generated in the blood driven through it. If the valve curtains are incompetent, a fluid vein is generated in the blood which leaks back. Murmurs are considered in more detail in connection with valvular disease.

Auscultation is also applied to the *bloodvessels*. A murmur is frequently heard over an aortic aneurysm. An aortic valvular murmur may be traced into the carotid artery. A continuous hum is sometimes heard over the large veins of the neck in anæmia; and a similar hum may be audible

over the left innominate vein at the manubrium sterni, in connection, it has been supposed, with enlargement of the bronchial glands.

5. ENDOCARDITIS.

Three varieties of endocarditis are commonly described: simple acute, ulcerative, and chronic. The second form is undoubtedly due to micro-organisms. In the first form, also, organisms are found, and these are of the same kind as in ulcerative endocarditis, the pyogenic cocci (streptococcus, staphylococcus, pneumococcus, and gonococcus) being most common, though occasionally the bacilli of enteric, tuberculosis, diphtheria, and anthrax have been met with. Some writers therefore are disposed to minimise the differences between the simple and the ulcerative varieties, but, though all gradations are met with between the two forms, they are, in typical cases, quite distinct both clinically and anatomically.

Simple Acute Endocarditis (Verrucose, Vegetative, or Benign Endocarditis).

Etiology.—This condition almost never occurs primarily. It is generally a manifestation of rheumatism, and is hence often associated with other rheumatic phenomena, such as polyarthritis, chorea, or pericarditis. It occasionally follows scarlet fever—another infection in which polyarthritis is sometimes observed. Measles, pneumonia and other fevers, tonsillitis, pregnancy, Bright's disease, and in rare instances diphtheria, may also be accompanied by simple endocarditis. Like rheumatism, it is most common in childhood and adolescence. The inflammation is usually confined to the valves and chordæ tendineæ on the left side of the heart, except in fœtal life, when the right heart suffers. These facts are explained by the special strain to which the left and right hearts are habitually exposed after and before birth respectively. The mitral valve suffers more frequently than the aortic.

Micro-organisms are often found in connection with the vegetations in this disease. The pyogenic cocci are most

common, but now and then others have been observed. It is possible that in some cases a chemical irritant in the blood is the cause of the inflammation.

Morbid Anatomy.—Numerous little warty projections or vegetations are produced along the lines of contact of the inflamed valve-segments. In the aortic valve, they are on the ventricular aspect, and in the mitral valve on the auricular aspect of the curtains. These warty vegetations consist mainly of fibrin, leucocytes and blood platelets, the fibrin being deposited by the blood-stream on the inflamed surface. They tend to undergo transformation into connective tissue. Some of these vegetations may become detached and give rise to embolism, but such emboli are small and seldom of much importance. The inflamed and softened valve, however, may yield to the blood pressure, so that an aneurysm develops which, by bursting, gives rise to perforation of the valve. If the inflammation should extend to the chordæ tendineæ, these may soften and rupture, and thus permit of regurgitation through the mitral orifice.

Symptoms.—Any symptoms that might be referable to this form of endocarditis are masked by those of the disease (*e.g.*, rheumatism) to which it is secondary. Continued pyrexia, with rapid action of the heart, after the subsidence of rheumatic polyarthritis, and also palpitation, have been suggested as evidence, but their value is extremely doubtful. We have to be guided by physical signs pointing to obstruction or regurgitation at the valvular orifices, and even then mitral regurgitation and its physical signs may be due to another cause than endocarditis. Certain evidence is often forthcoming later on, after chronic valvular disease has been established.

Diagnosis.—As a cardiac murmur is likely to be the sole evidence of endocarditis in the course of rheumatism, other possible explanations of the murmur must be borne in mind. It might be due to a former attack of endocarditis, and corroboration of this view might be found in signs of enlargement of the heart, and a history of rheumatism in the past. Or the murmur might be hæmic, a result of the blood state, in which case it would probably be widely diffused over the

cardiac area, and in any case more marked at the pulmonic area than elsewhere. A murmur due to actual regurgitation through the mitral orifice in consequence, not of endocarditis, but of stretching of the auriculo-ventricular orifice, can scarcely be distinguished from one due to endocarditis.

Prognosis.—Death rarely occurs directly from this disease, but very often chronic valvular disease remains, and ultimately leads to a fatal issue. On the other hand, both cardiac murmurs and cardiac symptoms which have followed acute rheumatism do occasionally, after years, pass away completely. Endocarditis may recur with a recurrence of the rheumatic infection. A damaged valve is specially liable to be attacked by ulcerative endocarditis.

Treatment.—Rest in bed is essential. The rheumatic attack should be cut short by treatment. With regard to the endocarditis, it is doubtful if anything does good except rest. This should be prolonged for several weeks, or perhaps months, after the fever subsides.

Ulcerative Endocarditis (Malignant or Infective Endocarditis).

Etiology.—While due to infection, this is not a specific disease like enteric fever or like Mediterranean fever, inasmuch as different cases may be due to different species of micro-organisms. The usual microbes are the *Streptococcus pyogenes*, the *Diplococcus pneumoniæ*, and the *Staphylococcus pyogenes aureus*, the two former each accounting, it is said, for about a third of the cases. Much less common are the gonococcus, the typhoid bacillus, and the tubercle bacillus. The disease is often associated with some other infection, such as pneumonia, puerperal fever, acute osteomyelitis, gonorrhœa, or a suppurating wound, which explains how the organism obtained access to the heart. Frequently, however, the source of the infection cannot be discovered. An important predisposing cause is a pre-existing lesion of the valve.

Morbid Anatomy.—It is chiefly the left side of the heart which suffers, though the right heart does not escape with the same proportionate frequency as in simple endocarditis. The valves suffer most, but the infection tends to spread to

other parts of the endocardium. The chordæ tendineæ may thus be softened and torn, and a mitral curtain, being thus permitted to flap upwards, may inoculate the wall of the auricle. An aneurysm may develop in a valve-segment and lead to perforation. Aneurysm or abscess may occur in the wall of the heart.

At the seat of lesion, there is round-celled infiltration with deposition of fibrin, which is often in great abundance. The micro-organisms are present in this fibrin. Portions of the latter are liable to be detached, and to give rise to embolism in the spleen, kidneys, brain, and other parts. In addition to the results of mere obstruction by the emboli, the organisms which they contain sometimes give rise to small metastatic abscesses, and, as the affected valve is generally on the left side of the heart, the condition then merits the name given it by Wilks, 'arterial pyæmia.' If the emboli come from the right heart, the metastatic abscesses will be chiefly in the lungs, as in ordinary pyæmia. An embolus may so damage the wall of a peripheral vessel that an aneurysm results.

Symptoms.—These are of two kinds, the one arising from the local condition in the heart, and the other from the septicæmia. The former include dyspnœa, dropsy, and the various phenomena met with in valvular disease, and are associated with physical signs of cardiac lesion. In particular there are apt to be murmurs, and these may change their character from time to time as the disease progresses. But on the whole, the cardiac or local phenomena are likely to be less urgent than the septic or general phenomena. The latter include an irregular temperature, rigors, sweating, enlargement of the spleen, and hæmorrhages into the skin, retinæ and mucous membranes. In addition there may be evidence of embolism of the kidney, as shown by the sudden onset of albuminuria, sometimes with hæmaturia and with pain in the back ; or of embolism of the spleen, as indicated by pain in the splenic region, splenic enlargement, and sudden elevation of temperature ; or, again, of embolism of the brain, as indicated by sudden hemiplegia, etc.

The course of the disease varies much in different cases, and the following types have been recognised, viz., septic, typhoid, pyæmic, cardiac and cerebral. The *cerebral* type may simulate meningitis, either of the tubercular or of the cerebro-spinal variety. Delirium, pyrexia, profuse sweating, hæmorrhagic and other skin eruptions, convulsions and coma are among the symptoms. As regards the heart, there may be no symptoms, and possibly no abnormal physical signs. Meningitis may actually be present.

To the *cardiac* type belong cases of chronic heart disease, in the course of which fever and septic symptoms supervene owing to the damaged valves becoming the seat of ulcerative endocarditis. These cases may be acute or chronic.

In the *pyæmic* type, there are multiple abscesses—*e.g.*, in the joints or in the lungs—but otherwise the cases need not be separated from those belonging to the septic type.

In the *septic* type (including the pyæmic), there is often a recognisable source of infection, such as a suppurating wound, acute osteomyelitis, or the puerperal state. The severe rigors occurring at irregular intervals, the wide ranges of temperature, the profuse sweating, the emaciation and the anæmia, with splenic enlargement, hæmorrhages, delirium, diarrhœa, and perhaps pulmonary symptoms and arthritis, point naturally to a septicæmic or pyæmic condition.

Osler calls attention to a particular variety of this type which simulates quotidian or tertian ague. It may develop without discoverable external cause in cases of chronic heart disease, and follow a chronic course.

The *typhoid* type is the most common of all, and may suggest a continued fever such as enteric. The temperature is not so irregular as in the septic type, and the general febrile symptoms may be the first sign of imperfect health. A dry tongue, delirium, early general prostration, free sweating, and diarrhœa with stools like those of enteric may also be present. There is often, but not always, a cardiac murmur. Stupor and coma may be present for a time before death.

Diagnosis.—This rests upon the combination of cardiac and septic phenomena, to which there are often added

evidences of multiple embolism. If these three groups of phenomena are present in an individual who has previously suffered from valvular disease and who has recently suffered from an infection, the evidence is very strong. Bacteriological examination of the blood during life will sometimes reveal the particular organism which is at fault.

The disease is apt to be mistaken for *enteric fever*. In the latter the temperature is less irregular, rigors seldom occur in the course of the disease, cardiac symptoms are absent, and Widal's reaction may be present. It is to be remembered, however, that pyæmia may develop in the course of enteric. In one case of this kind the *Staphylococcus aureus* was cultivated from the blood during life, as well as from the organs after death ; so that the detection of a pyogenic organism in the blood during life does not prove the existence of ulcerative endocarditis to the exclusion of enteric fever.

Malaria may be differentiated by microscopic and bacteriological examinations of the blood.

Pyæmia and ulcerative endocarditis are almost the same thing, and may be combined. The presence of an external wound, followed by general and pulmonary symptoms, and without signs of involvement of the heart, would suggest pyæmia proper ; whilst the presence of heart disease, to which general symptoms and multiple embolisms on the arterial side are added, would suggest ulcerative endocarditis.

Prognosis.—Death may take place within a few days, or may be postponed for many months. Only a few cases recover from the infection.

Treatment.—Any recognisable source of infection should be removed if possible. If it can be ascertained that the streptococcus is the organism concerned, a polyvalent antistreptococcic serum should be administered. Quinine, mercury, and arsenic may also be employed.

Chronic Endocarditis is often a sequel of the acute form, and is characterised chiefly by new formation of connective tissue. The process begins in the granulation tissue of the acute lesion, and tends slowly to spread to the remaining parts of the valves and the chordæ tendineæ. The valves thus become thickened, rigid and shrunken. Inflamed

valve-segments may coalesce. Lime-salts may be deposited in the new-formed connective tissue, so that the valves become brittle, and calcified portions may be broken off and give rise to embolism. Mitral stenosis sometimes develops in an insidious way without any evidence of preceding acute endocarditis.

Another cause of chronic endocarditis, especially at and after middle life, is high arterial tension such as results from gout, kidney disease, and habitual excess in eating and drinking. Occupations which involve a frequent though not continuous strain upon the vascular system also tend to induce it, as in blacksmiths, engineers and soldiers. The aortic valves suffer most in these cases.

Syphilis is a cause of chronic endocarditis before middle life, and senility at a later period. Occasionally an isolated area of endocarditis is found on the inner aspect of the auricle or ventricle, with or without disease of the valves, and the process may invade the myocardium.

The lesions of chronic endocarditis may at any stage become complicated by the occurrence of acute endocarditis, either in its simple or in its more dangerous ulcerative form.

6. VALVULAR DISEASE.

Etiology.—The most common cause of valvular disease is acute endocarditis, which is usually a result of the rheumatic infection. Except in the comparatively rare ulcerative endocarditis, serious harm is seldom done in the acute stage, but this is often followed by an extensive new formation of connective tissue which deforms the valve-structures. This is commonly known as chronic endocarditis, though the process has often been stationary for a long time before death. Sometimes, however, it is progressive.

The various forms of chronic endocarditis are causes of valvular disease, and are too often progressive. In rare instances, again, a valve—generally the aortic—is ruptured. This is usually the effect of severe muscular effort upon a valve already the seat of disease.

But apart from the conditions which give rise to actual deformity, a valve may become *relatively* incompetent, owing to the orifice which it ought to close becoming enlarged. This occasionally happens late in life at the aortic valve, and is quite common in old and young at the mitral orifice. The latter is surrounded by a muscular ring, which ought to contract when the ventricle contracts. If for any reason this muscle has been overstretched, or has lost its tone, it will be unable to narrow the orifice sufficiently, and the mitral curtains will not meet accurately.

Valves Affected.—Apart from tricuspid regurgitation, the lesions under consideration are almost confined to the left side of the heart. The most common single lesion is mitral incompetence ; mitral stenosis probably comes next ; then aortic incompetence ; and last of the series, aortic stenosis. The most common combined lesions are mitral incompetence and stenosis ; aortic and mitral incompetence ; and aortic stenosis and incompetence. A still more formidable combination consists of stenosis and incompetence of the mitral, tricuspid, and aortic valves.[1]

Tricuspid incompetence is very common, and by far the most common right-sided lesion, but it is almost always of a relative kind, and secondary to disease on the left side of the heart, or to disease of the respiratory system.

When the normal current of blood through a valve of the heart is obstructed by narrowing of the orifice, there is said to be ' stenosis ' or ' constriction ' of that valvular orifice, and the result is ' obstruction ' at that valve. On the other hand, if the valve is unable to close the orifice sufficiently to prevent a leakage in the wrong direction, there is ' incompetence ' or ' insufficiency ' of, and ' regurgitation ' at, that particular valve.

Cardiac Murmurs.—These vary greatly in their duration, loudness, harshness and sundry other properties which are not of very great service in diagnosis. The important facts with regard to a murmur, and those which give the clue to

[1] I have met with this combination in a case where there was also an old abscess in the heart wall, containing pneumococci (*Trans. Glasg. Path. and Clin. Soc.*, January, 1900).

the valve-lesion which produces it, are (1) the rhythm, (2) the seat of maximum intensity, and (3) the direction of propagation.

Murmurs which correspond in rhythm with the systole of the ventricle are known as systolic, or ventricular systolic (V.S.). Those which coincide in time with the diastole of the ventricle are called diastolic, or ventricular diastolic (V.D.). Those which correspond in time to the contraction of the auricle are called presystolic, or auricular systolic (A.S.). Murmurs occurring in the long pause are sometimes distinguished as early diastolic, mid-diastolic (mesodiastolic), late diastolic, and entire diastolic. Late diastolic is practically the same thing as presystolic or auricular systolic.

The relations between the different valves, lesions and murmurs are shown thus :

Valve.	Lesion.	Murmur.
Aortic, Pulmonic	Stenosis	Systolic (V.S.).
Mitral, Tricuspid	Incompetence	Systolic (V.S.).
Aortic, Pulmonic	Incompetence	Diastolic (V.D.).
Mitral, Tricuspid	Stenosis[1]	Presystolic (A.S.).

Thrills, as recognised by palpation, are named according to their rhythm in the same way as murmurs.

Mitral Incompetence.

Etiology.—Mitral incompetence may result from acute or chronic endocarditis, which causes deformity or rigidity of the valves and chordæ tendineæ. The consequence is that the curtains of the valve do not meet accurately when the ventricle contracts. *Relative incompetence* results from many conditions which cause dilatation of the left ventricle, or loss of tone of the muscular ring surrounding the orifice.

[1] Mitral or tricuspid stenosis may be associated with an early, middle, or entire diastolic, instead of a late diastolic (presystolic) murmur.

Among these conditions are anæmia, acute disease, the debility of advancing years, persistent high arterial tension, and aortic valve disease.

Morbid Anatomy.—As some blood regurgitates into the auricle during the ventricular systole, the auricle becomes dilated so as to accommodate this quantity in addition to that which is normal. The auricle at its systole drives this increased quantity into the ventricle, which thus becomes dilated so as to contain the quantity it drives into the aorta and also the quantity which escapes back into the auricle. The auricle and ventricle will naturally undergo some hypertrophy as well as dilatation. The increased pressure in the left auricle, due to the regurgitation, causes increased resistance to the outflow from the pulmonary veins, and accordingly the tension in the pulmonary system is raised. This throws increased work upon the right ventricle, which in its turn becomes dilated and hypertrophied.

This condition of matters may persist indefinitely, compensation being complete ; but if the mitral lesion should be progressive and not stationary, or if the general health should fail, then the right ventricle is apt to yield, and relative incompetence of the tricuspid valve ensues.

In cases of long standing, there is brown induration of the lungs, which is characterised by dilatation of capillaries, hæmorrhage by diapedesis, pigmentation, and increase of connective tissue. Under the same circumstances, atheroma of the pulmonary artery is sometimes observed.

Symptoms.—If good compensation exists, there may be no symptoms at all ; or the individual may become breathless after exertion more readily than a perfectly normal person, though otherwise he is quite free from symptoms. The early symptoms of the disease point to obstruction in the pulmonary vessels ; but at a later stage, when the right ventricle fails, engorgement of the systemic veins and dropsy are important phenomena. There are shortness of breath, aggravated by exertion ; cough ; sometimes hæmoptysis ; palpitation ; and lividity of the surface, with or without pallor. As the condition becomes worse, dyspnœa becomes severe and amounts to orthopnœa ; the liver enlarges ; the

lungs become œdematous; the urine is scanty and concentrated; dropsy begins in the dependent parts and extends; effusions accumulate in the serous cavities; quiet delirium sets in, and sleep is almost impossible. The distress may be aggravated by the occurrence of pulmonary embolism, as shown by hæmoptysis, pain in the side, pleural friction-sound and signs of localised consolidation.

Physical Signs.—The *murmur* is systolic (V.S.) in rhythm. Its maximum intensity is at the apex. Its direction of conduction is outwards from the apex into the lateral region of the chest. It is often heard at the back about the lower angle of the scapula, and it may actually be louder there than in the lateral region. The murmur is probably conducted to the back partly by way of the ribs, and partly by way of the base of the ventricle and the spine.

The murmur may simply suggest a prolongation of the first sound. Or murmur and first sound may both be distinct and accompany one another. Or, again, the murmur may actually take the place of the first sound, the latter being imperceptible. The murmur may be soft or harsh, and is sometimes musical. It is occasionally accompanied by a thrill of the same rhythm.

The pulmonic second sound is probably accentuated, and as long as this is the case, the right ventricle is doing good work.

Unless the lesion is slight, the visible area of cardiac *pulsation* is increased. The apex beat is displaced downwards from enlargement of the left ventricle, and to the left owing to enlargement of both ventricles.

The area of *cardiac dulness* is increased horizontally to right and left owing to enlargement of both ventricles.

The *pulse*, unless in slight cases, tends to be irregular, both in force and in rhythm (Fig. 25).

Signs of free regurgitation include the following: (1) complete replacement of the first sound by the murmur, suggesting loss of mitral valve tension; (2) accentuated pulmonic second sound; (3) enlargement of the left side of the heart; (4) enlargement of the right heart; (5) a small, weak pulse conjoined with a strongly acting ventricle,

suggesting that much of the blood dealt with by the left ventricle goes elsewhere than into the aorta. Nevertheless, with almost all, and perhaps with all of these conditions present, compensation may be good in the absence of unusual strain.

The absence of the above signs (if they have had time to develop), the absence of symptoms, a murmur which follows or accompanies a well-marked first sound, and perfect regularity of the pulse, indicate that the lesion is slight.

Prognosis.—Mitral regurgitation is the least formidable of the valvular affections. When the lesion is slight, the patient may lead a life of hard work and live to advanced age, without any inconvenience as regards his heart. In

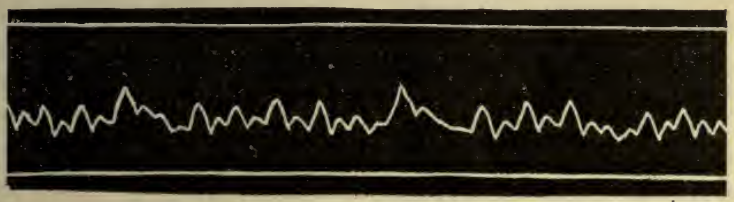

FIG. 25.—PULSE OF MITRAL REGURGITATION.

some cases, indeed, where both symptoms and physical signs of cardiac disease have followed acute rheumatism, both symptoms and signs disappear after the lapse of years. Where the lesion is more severe, the patient may be seriously disabled from time to time by temporary failure of compensation (*e.g.*, owing to overwork or pregnancy), and yet may recover under treatment and survive for many years. Thus a patient who recovered in the Glasgow Royal Infirmary from severe failure of compensation in mitral disease had been in hospital twenty-three years previously, when dilatation of the heart and presystolic and systolic mitral murmurs were noted.

When mitral regurgitation is due to dilatation of the ventricle in consequence of high arterial tension or aortic disease, the prognosis, generally speaking, is not so favourable. On the other hand, when the dilatation occurs in

connection with acute disease or anæmia, recovery should be complete, provided that the primary disease is cured, and the patient takes sufficient rest.

Mitral regurgitation is not likely to cause sudden death.

Treatment.—In cases of anæmia and debility, the primary disease must be treated, and for the heart itself prolonged rest is necessary, after which gentle exercises should be gradually introduced. In cases occurring in elderly people, with no symptoms of cardiac failure, regular exercise is important, and great moderation in eating and drinking must be observed. The bowels must be kept regular, and if high tension is present, a small dose of mercury should be given twice a week. All mitral cases should be carefully guarded against chills, which by attacking the respiratory organs might impair compensation.

When distinct cardiac symptoms are present, rest in bed is essential, with a light, spare diet and good nursing. A mercurial purge (*e.g.*, pil. hydrarg. et pil. colocynth. et hyoscyam., āā gr. v.) should be given at once, and repeated from time to time. In some cases it may be well to spare the heart by restricting the fluids taken as well as the solids. The most important drug is digitalis, which may be given in various ways ; a good formula is the well-known pill containing powdered digitalis, powdered squill, and calomel (or blue pill), of each 1 grain, to be given three or four times a day. If the liver is enlarged and tender, Broadbent's excellent plan of applying several leeches over that organ may be adopted. This is a very gentle mode of blood-letting, and will probably relieve or remove local pain ; but in young subjects much larger quantities of blood may be withdrawn by venesection if the dyspnœa and cyanosis are great. Effusions in the pleural cavities should be removed by the aspirator, and great swelling of the legs and ascites should be dealt with by Southey's tubes. Removal of dropsies by one or other of these means sometimes brings about very speedy relief. It naturally removes much pressure from the blood-vessels, and thus eases the task of the overburdened heart. Strychnine ($\frac{1}{60}$ to $\frac{1}{30}$ grain hypodermically every four, six, or

eight hours) and ammonium carbonate are valuable cardiac stimulants.[1]

The patient often suffers terribly from want of sleep, whereas it is very important that he should sleep well. Paraldehyde in doses of 1 to 2 drachms frequently repeated is safe, but not always effectual. Morphine is sometimes of immense benefit, while in other cases it will increase the cyanosis. If the urine is not very scanty, and if the dyspnœa and insomnia seem greater than can be accounted for by the physical signs in the respiratory system (and especially signs of pulmonary œdema or bronchitis), morphine should be tried hypodermically, beginning with a very small dose ($\frac{1}{10}$ to $\frac{1}{8}$ grain).

When the case is responding to treatment, two signs speedily show it—the patient rests further back in bed, and the urine increases in quantity. Sometimes digitalis does not agree—possibly because it has already been pushed before the patient comes into hospital. The signs of disagreement are sickness and increased scantiness of urine. In such a case, strophanthus and strychnine may be given instead.

As recovery takes place, the aim should be to replace digitalis by tonics, such as iron, arsenic, and quinine. Strychnine should be continued in reduced quantity. The patient should rest in bed for six or eight weeks, then get up to the couch for a part of the day, and then begin gentle walking exercise with great caution.

Mitral Stenosis.

Etiology.—Stenosis of the mitral orifice results from endocarditis, and is generally of rheumatic origin. It commences chiefly in later childhood and early adult life, and is often of very insidious onset. Frequently there is no history of arthritis or of any other symptom which the patient regarded as rheumatic. Mitral stenosis is much more common in

[1] Alcohol should be avoided in these cases as a rule. Wilks pointed out many years ago that its influence is harmful (*Brit. Med. Jour.*, 1891, ii. 462. See also Monro and Findlay, *Glasg. Med. Jour.*, 1904, lxi., pp. 329-340).

females than in males, and whilst it doubtless generally commences in later childhood, the patient generally comes under the notice of the physician when aged from fifteen to twenty-five years.[1] Broadbent suggests that the high tension which results from anæmia is a cause. Gout, pregnancy, and chronic kidney disease also favour the development of mitral stenosis.

Morbid Anatomy.—In pure mitral stenosis, the left ventricle is not enlarged. The left auricle and right ventricle are dilated and hypertrophied. In well-marked cases, the mitral orifice is so narrowed that it will just admit the tip of the finger, and yet in such a case the patient may have been free from well-marked cardiac symptoms till a day or two before death. The valves are thickened and rigid, and their chordæ tendineæ are shortened. The curtains are often welded together, and the orifice, viewed through the auricle, is seen as a mere slit, the 'button-hole' mitral—the form usually seen in adults. In children, the valve structures are usually transformed into a hollow truncated cone—the 'funnel mitral.' As the left auricle has but a thin muscular wall, compensation in this disease depends mainly upon the right ventricle.

Symptoms.—Symptoms may be quite absent, or there may be shortness of breath on exertion, perhaps with hæmoptysis. Indeed, of the different valvular diseases, mitral stenosis is the one in which free and repeated spitting of blood is most common. This is due to the high and increasing tension of blood in the pulmonary vessels. The ends of the fingers are clubbed in some cases. Embolisms may occur in the brain, spleen, and kidneys, owing to detachment of portions of thrombus from the dilated left auricle. In rare instances paralysis of the left vocal cord is caused by the enlarged auricle compressing the left recurrent nerve.

When compensation fails, the symptoms are in the main similar to those of mitral regurgitation, but dyspnœa is much more obtrusive than dropsy, and the latter is often absent. Broadbent points out that this form of valvular disease is specially apt to be associated with pulsation of an enlarged

[1] See footnote on p. 95.

liver, and that ascites may develop even before œdema of the feet. Indeed, even on her death-bed, the patient may present almost no symptoms except general weakness and a little shortness of breath, with a very small pulse. Distinctive physical signs as well as marked symptoms may be absent.

Physical Signs.—The characteristic *murmur* is presystolic (A.S.) in rhythm. It is rough in character, and increases in intensity up to the first sound, when it suddenly stops. Its usual area of audibility is restricted to the apex region and immediately internal to and above it, so that the direction of propagation scarcely needs to be considered. Occasionally, however, an A.S. murmur is found to be widely distributed over the chest.

Other murmurs of a less characteristic kind may be audible in the diastolic period in cases of mitral stenosis, and it is important not to confuse them with aortic regurgitant murmurs. These are the early diastolic, mid-diastolic, and entire diastolic murmurs. The first is to be accounted for by the elastic recoil of the over-distended pulmonary veins and left auricle. The mid-diastolic is not so easily explained unless it be due to the suction power of the left ventricle. The typical presystolic or late diastolic murmur is caused by the contraction of the auricle. The entire diastolic murmur, occupying the whole of the long pause, must be attributed to these different causes acting in succession.

The pulmonic second sound is accentuated, and in a considerable proportion of cases is reduplicated. Reduplication of the second sound may be the first sign of commencing mitral stenosis.

The apex beat is little if at all displaced in pure mitral stenosis, because the left ventricle is not enlarged. Great enlargement of the right ventricle may, however, push the left ventricle slightly towards the left. The impulse is diffuse, and the area of dulness is increased towards the right, owing to the enlargement of the right ventricle.

The A.S. murmur of mitral stenosis is often accompanied by an A.S. *thrill*. Sometimes there is a thrill without a murmur ; or, again, neither may be recognisable.

The *pulse* is usually regular until late in the disease.

Its special features are small volume and high tension (Fig. 26).

Broadbent recognises three *stages* in the disease, distinguished by auscultation. In the *first*, the presystolic murmur, the first sound and the second sound are all audible at the apex. In this stage there are no serious symptoms.

In the *second* stage, the murmur remains audible, but the first sound becomes short and sharp like a normal second sound, and the second sound at the apex is lost. The murmur should be carefully timed by placing the finger over the apex impulse, or, failing that, the carotid artery. The loss of the second sound (probably aortic second sound) at the apex is accounted for, first, by the greatly enlarged right ventricle overlapping the left and causing it to be withdrawn from the chest-wall ; and, secondly, by the small volume of

Fig. 26.—Pulse of Mitral Stenosis, Early Stage.

blood passed by the ventricle into the aorta being insufficient to cause any great recoil. The change in the character of the first sound is not so well understood. The element of duration is lost ; the element of tension remains. Broadbent suggests that the left ventricle contracts before it has been completely filled through the narrowed mitral orifice. Its contraction is at first unresisted, but as soon as its cavity is reduced sufficiently to be filled by the contained blood, its walls suddenly become tense. This explains the loudness of the sound, and as the actual working part of the systole is shortened, so, it is suggested, the first sound may be correspondingly shortened. Sansom thinks the peculiar first sound is due to sudden contraction of the *right* ventricle, which acts with abnormal force against abnormal resistance.

In the second stage, there may be few or no serious symptoms if the patient lives a quiet life.

In the *third* stage, the murmur also disappears, and only the short and sharp first sound persists at the apex. This third stage is attributable to failure of the right ventricle and tricuspid regurgitation, so that the blood is not driven through the mitral orifice with sufficient force to generate a murmur. This stage is usually accompanied by serious symptoms, but under treatment the patient may regain the second stage.

The three stages described cannot always be recognised. Thus, the second sound may be quite distinct at the apex when death is approaching.

Prognosis.—Mitral stenosis is not so dangerous as aortic incompetence, and does not cause sudden death in the absence of serious symptoms. The latter may, however, extend over only a few days. As compared with mitral incompetence, there is the serious fact that mitral stenosis is often a progressive lesion. Nevertheless, patients often survive for many years in fair health, though handicapped by shortness of breath ; and they may recover over and over again from cardiac disablement with severe symptoms.

Treatment.—In the early stages when no symptoms are present, active treatment is not required, but the patient should guard against chills, so as to avoid rheumatism and bronchitis, and should guard against high arterial tension by the avoidance of over-eating, over-drinking, and constipation.

When symptoms are present, rest and mercurial purges are the most important remedies. If the symptoms are urgent, and include orthopnœa, enlargement of the liver, and dropsy, mercurial purges should be promptly put in action, and it is sometimes desirable to practise venesection, though in other cases leeching is sufficient. After thorough purging, if the right ventricle is still very feeble, digitalis may be given in a tentative way for a time, with ammonium carbonate ; but as soon as practicable, these stimulants should be replaced by tonics like strychnine, to which one of the nitrites may be added to keep down arterial tension.

Aortic Stenosis.

Etiology.—This condition may result from thickening and rigidity of the valve segments in consequence of chronic inflammatory and degenerative processes, especially in later life. Or it may be due to the endocarditis of rheumatism, scarlet fever, etc.; or occasionally to syphilis. The valve segments may be partly adherent to one another, and recent vegetations may increase the obstruction to the blood-flow. Such changes in the valves are very apt to cause incompetence at the same time as stenosis. Pure aortic stenosis is less common than aortic incompetence, and neither is so common singly as a combination of the two. In rheumatic cases, the mitral valve is usually diseased at the same time.

Morbid Anatomy.—In addition to the changes at the aortic valve, there is hypertrophy of the left ventricle. In pure aortic stenosis, this hypertrophy is pure—*i.e.*, not accompanied by dilatation. But if compensation ultimately fails, the ventricle dilates, and this leads to secondary mitral incompetence, with the associated changes in the left auricle, lungs and right heart.

Symptoms.—These may be quite absent for a long time. When compensation fails, the patient may suffer from pallor, muscular weakness, giddiness and faintness, these symptoms being explained by insufficiency of the blood-supply to the skin, muscles and brain. As the ventricle continues to fail, the mitral valve becomes incompetent through dilatation of the ventricle, stretching of the curtains and chordæ tendineæ, and sometimes by rupture of the two latter. Symptoms referable to mitral regurgitation naturally follow.

Physical Signs.—The *murmur* is systolic (V.S.) in rhythm. It is heard with greatest intensity at or near the aortic cartilage, where it may be associated with thrill. It is conducted upwards to the manubrium and right sterno-clavicular joint, to the suprasternal fossa and into the carotid arteries. It is sometimes conducted by the descending aorta to the spine, and it may be conducted by the left

ventricle to the apex. In the latter case, it may be inaudible over the right ventricle.

It is important to remember that without any aortic stenosis, a murmur may be heard at the aortic cartilage (1) in connection with anæmia and acute fevers (though such hæmic murmurs are usually heard better at the pulmonic area); (2) in consequence of mere roughening of the aortic orifice; and (3) in consequence of dilatation of the aorta beyond the orifice.[1] Such causes of murmur will be recognised by the history of the case and by the accompanying symptoms and physical signs.

In aortic stenosis, the apex is displaced downwards and slightly outwards. As long as compensation is maintained, the impulse is deliberate, the first sound at the apex is prolonged, but not loud, and the aortic second sound is not well marked.

The *pulse* is of small volume, and the tracing shows a gradual rise and a rounded summit. Without the conditions of pulse and heart just described, a V.S. aortic murmur is no evidence of actual stenosis of the aortic orifice.

The *severity of the lesion* is to be estimated by the degree of modification of the pulse and hypertrophy of the heart.

Prognosis.—This disease is more dangerous than mitral incompetence, but less dangerous than aortic incompetence, and probably also mitral stenosis. It does not involve the risk of sudden death. When distinct symptoms have set in as a result of secondary mitral incompetence, the outlook is grave.

Treatment.—Syphilitic cases require iodide of potassium. In the absence of symptoms, the principal thing is to avoid excesses in food, drink and muscular exertion. Constipation must be avoided. When the ventricle fails, digitalis may be given to strengthen the heart, with nitrites to relax the arterioles. If the symptoms pass away, the patient should

[1] When the aortic valve is normal, but the aorta immediately beyond it is dilated, the condition has been spoken of as *relative aortic stenosis*, but this is not to be regarded as in any way a disease of the aortic valve, although it may give rise to a systolic murmur close to the area of aortic murmurs.

continue to rest for some weeks, and then gradually begin to move about. At this stage, arsenic, iron, quinine, and strychnine may be prescribed.

Aortic Incompetence.

Etiology.—The causes are ulcerative and simple acute endocarditis, more commonly chronic endocarditis or atheroma, occasionally syphilis, and in rare instances rupture of a valve segment. Sometimes the incompetence is *relative*, being due to stretching of the orifice. Syphilis should be suspected if the lesion sets in before the degenerative period of life in a case where there is no evidence or history of rheumatism, high arterial tension, or other possible cause of chronic endocarditis, and especially if the symptoms develop subacutely. Syphilis may also, along with alcoholism and frequent strain, be regarded as a cause of atheroma.

Morbid Anatomy.—There may be great deformity and rigidity of the valve segments. Frequently the neighbouring part of the aorta and the coronary arteries are the seat of atheroma. Stenosis is sometimes present, especially in cases resulting from acute endocarditis. The left ventricle becomes dilated and hypertrophied, so that the heart may be of enormous size. Aortic regurgitation yields the greatest enlargement of the heart, the *cor bovinum*. Secondary incompetence of the mitral valve with enlargement of the left auricle and right ventricle may be present.

Symptoms.—These differ greatly from the symptoms of mitral disease. They include pallor, faintness, muscular weakness, oppression in the chest, dyspnœa on exertion, paroxysmal dyspnœa, disturbed sleep with bad dreams, and sometimes pain in the cardiac region or pain more widely distributed as in angina pectoris. Vomiting may occur, and is of bad omen. Mental symptoms may be present, and occasionally even a suicidal tendency. Dropsy, cyanosis and pulmonary congestion are seldom conspicuous, and are usually absent till a late stage.

Sudden death from syncope is common. In other cases,

death takes place from exhaustion which results from want of sleep, the struggle for breath, pain, and inability to take nourishment. A third group of cases is constituted by those in which secondary mitral regurgitation brings about the train of symptoms seen in mitral disease with broken-down compensation.

Physical Signs.—The *murmur* is diastolic (V.D.) in rhythm. It is usually audible at the aortic cartilage and the upper end of the sternum. It is conducted downwards, towards the lower end of the sternum, and also towards the apex. It is often heard much better at the lower end of the sternum than at the aortic cartilage, where indeed it may be quite inaudible. It is often faintly audible at the apex. The murmur is usually soft and might often be missed by a careless observer. It is occasionally musical. When the diastolic murmur replaces instead of following or accompanying the second sound, a considerable degree of incompetence exists. A double murmur can sometimes be heard in the femoral artery (' Duroziez's murmur ').

In this disease, a presystolic murmur (' Flint's murmur '), is sometimes heard at the apex ; it is probably due to the regurgitating blood falling upon the anterior curtain of the mitral valve, and thus causing a certain amount of mitral obstruction.

Other important signs are present, and the extent to which they are developed indicates in some measure the degree of severity of the lesion. Thus the apex is much displaced downwards and to the left ; the pulsation of the ventricle is diffuse ; and the area of cardiac dulness is greatly increased to the left and downwards. There is exaggerated pulsation of the arteries as seen in the neck and limbs. Capillary pulsation is seen in the form of alternating increase and decrease of redness—*e.g.*, in the skin of the forehead after it has been reddened by stroking with the finger-nail.

It has been commonly taught that the *pulse* as examined at the wrist is delayed, but Mackenzie's careful investigations by graphic methods seem to prove conclusively that this is erroneous. The artery is of large volume, doubtless

owing to the sudden distensions to which it is constantly being subjected. When the limb is elevated, the vessel is found to fill suddenly, but to be empty between the beats, owing to the leakage at the aortic valve (collapsing or Corrigan's pulse, Fig. 27). If, however, aortic stenosis coexists, or if the aorta and other vessels are inelastic owing to degenerative changes, the characteristic pulse may not be apparent (Fig. 18, p. 265).

The *severity of the lesion* is to be judged by the degree of enlargement of the heart, pulsation of the carotids, collapse of the pulse, capillary pulsation and pallor of the surface. If the murmur replaces the second sound, and does not simply accompany or follow it, free leakage is indicated.

Prognosis.—In cases where the lesion results from the acute endocarditis of early life, compensation may remain

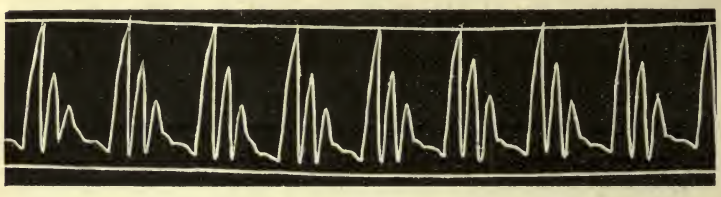

Fig. 27.—Pulse of Aortic Regurgitation.

satisfactory and the patient may work hard for an indefinite period, provided that he rests sufficiently long at the outset for the necessary hypertrophy to take place. In late life the outlook is very different, for here the aortic lesion tends to be progressive, and to be associated with important changes in the aorta and coronary arteries, while hypertrophy is not likely to be sufficient for compensation. In very severe aortic incompetence, the supervention of stenosis in addition occasionally produces highly beneficial results.

Aortic incompetence is the only valvular disease which involves the risk of sudden death.

Treatment.—The patient should rest for some months after the lesion sets in, and such a lesion should be carefully watched for in children who present any of the rheumatic series of phenomena, however slight these may be. Moreover, prolonged rest should be enforced after any acute

intercurrent illness. When the patient is able to go about, he should avoid over-exertion, sudden efforts and mental excitement. He should never rise from the recumbent posture quickly, and particularly from a warm bed to pass water. The bowels must be carefully regulated, and an occasional course of iron may be advisable.

When the left ventricle fails, strychnine, atropine and ammonia should be given, and sleep should be procured, if need be, by hypodermic injections of morphine. Digitalis is contra-indicated as prolonging the diastole and increasing the risk of fatal syncope. Arsenic is a valuable tonic, and may be combined with potassium iodide. Broadbent recommends phosphorus. If angina pectoris is present, the appropriate remedies must be used.

If secondary mitral incompetence sets in, some of the milder measures appropriate to that condition may be employed—*e.g.*, mercurial purges. Digitalis is now allowable, but should not be continued longer than is required.

Valvular Disease of the Right Side of the Heart.

TRICUSPID INCOMPETENCE is usually relative. In rare instances, however, it may result from acute endocarditis occurring before or after birth, or from a chronic endocarditis due to constant excessive strain on the valve when the tension in the pulmonary circulation is abnormally high. Relative incompetence results from obstruction to the pulmonary circulation, leading to dilatation of the right ventricle, as in disease of the left side of the heart, bronchitis, emphysema, etc. It also occurs in healthy persons after violent exertion.

The *murmur* is systolic in rhythm (V.S.). Its maximum intensity is at and to the left of the left sternal margin, at the level of the apex. It is sometimes audible as far out as the apex. Its usual area may be described as corresponding to the right ventricle, and extending from the lower sternum to near the apex, and from the third to the sixth cartilage. There is no well-recognised direction of propagation beyond the limits of the right ventricle. A murmur is by no means

constant in tricuspid regurgitation, and in practice a tricuspid murmur is less important than the other evidences of leakage at the valve.

Tricuspid and mitral murmurs may coexist. The two may be distinguished in some cases by a difference of quality when they are examined at their special seats of maximum intensity. Moreover, the tricuspid murmur is not conducted round the left side of the chest. Similarly tricuspid and aortic or pulmonic murmurs may be distinguished by differences of quality, and by the fact that tricuspid murmurs are usually inaudible above the third cartilages. Concomitant signs and symptoms may throw light on the matter.

Two other signs, if present, are certain evidence of tricuspid regurgitation, viz., pulsation of the veins of the neck, and pulsation of the liver. Pulsation communicated from the carotid arteries must not be mistaken for actual pulsation in the veins, which is communicated from the right ventricle through the auricle and superior vena cava. The jugular veins may be greatly distended. Pulsation of the enlarged liver, which is recognised by placing one hand in front over the fifth and sixth costal cartilages, and the other at the right side of the chest over the lower ribs, is due to the ventricular pulsation being communicated by way of the inferior vena cava to the hepatic veins.

Other signs which may be noted, particularly if the incompetence is secondary to disease of the left heart, are: extensive heaving of the right ventricle in the epigastrium, enlargement of the right side of the heart, and pulsation of the heart to the right of the sternum.

In serious failure of the right ventricle, tricuspid regurgitation naturally leads to passive hyperæmia over the body generally, with enlargement and tenderness of the liver, albuminuria, etc. General dropsy is frequently but not invariably present.

The *prognosis* depends upon the condition to which the tricuspid regurgitation is secondary.

The *treatment* for failure of the right ventricle is that which has been recommended for mitral regurgitation with failure of compensation.

TRICUSPID STENOSIS may be congenital or acquired. The latter form occurs chiefly in adult females, and mitral stenosis is generally present in addition. In some cases the two valves seem to suffer from the same attack of endocarditis ; in others, the great strain on the tricuspid, resulting from the condition of the pulmonary circulation in mitral stenosis, appears to induce chronic inflammation of the tricuspid. The characteristic *murmur* is presystolic (A.S.), and is heard in the tricuspid area, but sometimes it cannot be recognised. An important sign is distension of the jugular veins without pulsation. When tricuspid stenosis complicates mitral stenosis, dropsy is likely to set in much earlier than in pure mitral stenosis.

PULMONIC STENOSIS is almost always congenital, and is often associated with other cardiac abnormalities. There may be a rough systolic *murmur* best heard to the left of the upper part of the sternum. The right heart is often hypertrophied.

PULMONIC INCOMPETENCE occasionally results from congenital malformation and from ulcerative endocarditis ; possibly also from excessive tension in the pulmonary system stretching the orifice of the valve. A diastolic *murmur* is heard in the pulmonic area, and is conducted downwards. Aortic regurgitation must be excluded by noting the characters of the aortic sounds, the pulsation of the carotids, the pulse-tracing, and the condition of the left ventricle. No special symptoms are recognised as due to pulmonic regurgitation.

7. CONGENITAL HEART DISEASE.

Congenital heart disease may be due to errors in development, or to fœtal endocarditis, or to both. Endocarditis affects chiefly the right side of the heart, and is of a chronic or sclerotic type. Moreover, a developmental flaw in a valve seems to predispose that valve to endocarditis.

In cases that are capable of extra-uterine existence, the principal changes are defects in the cardiac septa, stenosis in the region of the pulmonary orifice, and patency of the

ductus arteriosus Botalli. The most common is *stenosis of the pulmonary orifice*, which may be due to disease or malformation of the valve segments, or to narrowing of the trunk of the vessel, or to narrowing of the infundibulum (*conus arteriosus*) of the right ventricle. In some cases there is complete obliteration or atresia of the orifice. *Patency of the foramen ovale* may occur along with, and as a consequence of, pulmonic stenosis, which raises the pressure in the right heart. Or it may occur as an isolated anomaly. It does not necessarily give rise to symptoms. *Defect of the interventricular septum* is observed chiefly at the undefended space in the upper part of the septum, and is usually associated with some abnormality such as pulmonic stenosis, which causes increased pressure in the right ventricle. In such a case blood passes through the gap into the left ventricle. *Patency of the ductus arteriosus* (which conveys the blood in the fœtus from the commencement of the pulmonary artery to the descending aorta) may result from obstruction to the pulmonary circulation—*e.g.*, in consequence of pulmonic stenosis, which may at the same time cause patency of the foramen ovale.

Symptoms.—In a large proportion of cases there is a striking degree of cyanosis ; whence the expression *morbus cæruleus*, or ' blue disease,' which is practically equivalent to congenital heart disease. The blueness is specially noticeable in the nose, lips, ears, hands, and feet. The explanation of this condition is still obscure, but it is not simply the mechanical admixture of arterial and venous blood. The surface of the body is cold, and the ends of the digits are clubbed. The blood is concentrated, the red corpuscles and hæmoglobin being both in excess of the normal. The cyanosis may be noticeable only on exertion, but shortness of breath is in most cases constantly present.

Physical Signs.—In *pulmonic stenosis*, the right ventricle can sometimes be recognised as hypertrophied, and a rough systolic murmur is heard, sometimes over a wide area, but especially to the left of the upper part of the sternum in the pulmonic area. *Deficiency of the interventricular septum* is associated, according to Broadbent, with a quite character-

istic murmur which goes on without ceasing. It is harsh and loud, and becomes higher pitched and louder at the systole. *Patency of the foramen ovale* is not recognisable by any known physical sign. It may or may not cause a murmur, and if it does, the murmur may or may not be systolic. *Patency of the ductus arteriosus* is suggested by a prolonged thrill and murmur at the base, if there are no signs of cardiac hypertrophy. In most cases, however, of congenital heart disease, it is impossible to be certain as to the exact lesion.

Diagnosis.—The principal points are the cyanosis and continuous or paroxysmal dyspnœa dating from infancy, the clubbing of the digits, the murmur, and the cardiac hypertrophy.

Prognosis.—This is always unfavourable, though a few cases reach adult and even advanced life. Patency of the foramen ovale is not so serious as defect of the interventricular septum, or stenosis of the pulmonary artery Stenosis of the pulmonary artery seems to predispose to tuberculosis of the lungs. Infants with these malformations often die of convulsions. Children who survive for a time are apt to be stunted, both in mind and in body.

Treatment.—Patients of this class must be carefully clad and protected from undue exposure. When compensation fails, the treatment is similar to that of acquired heart disease.

8. HYPERTROPHY AND DILATATION.

Hypertrophy of the heart is recognised by increase in the thickness of its muscular wall ; dilatation by increase in the capacity of its cavities. The two conditions are often present together, and may involve one or several of the chambers of the heart (partial hypertrophy), or the whole organ (general hypertrophy).

Hypertrophy.

Hypertrophy is almost invariably a conservative process, and in view of the circumstances under which it occurs, is generally to be regarded with satisfaction.

Etiology.—Hypertrophy is principally seen in the ventricles when their muscular tissue becomes increased to enable them to overtake any constant or frequently recurring increase of work.

Thus, the *left ventricle* becomes hypertrophied in consequence of aortic obstruction or regurgitation, mitral regurgitation, rigidity of the arteries, high arterial tension, adherent pericardium, damage to the myocardium from disease of the coronary arteries, or prolonged and severe muscular effort. The last-mentioned form occurs in athletes, soldiers, and others, and used to be spoken of as *idiopathic hypertrophy*, because after death there is no obvious cause for it.

The *right ventricle* becomes hypertrophied as a result of mitral stenosis or incompetence, pulmonic stenosis, adherent pericardium, or disease of the respiratory system, such as emphysema or bronchitis, where there is increased resistance to the flow of blood through the pulmonary vessels.

The *left auricle* is hypertrophied, and at the same time dilated, in mitral disease, especially stenosis.

The *right auricle* becomes hypertrophied and dilated in tricuspid stenosis, and in conditions which cause hypertrophy of the right ventricle.

Morbid Anatomy.—When the *left ventricle* is much enlarged, the heart seems lengthened, owing to the apex projecting further downwards and to the left. When the organ is viewed from the front, the septum seems to be placed more to the right than normally, and on section it is seen to bulge into the right ventricle, whose capacity it may thus diminish. Hypertrophy of the *right ventricle* gives the heart a somewhat square shape, the apex being blunted, and the septum nearer the left border than normally. The weight of the heart is increased in hypertrophy, and is often twice the normal.

Symptoms.—It is scarcely fair to speak of symptoms as due to hypertrophy. Some that are described are the result rather of insufficient hypertrophy, or of the cause of the hypertrophy. There may, however, be discomfort from the widespread impulse, or tenderness at the apex, or irregu-

larity or undue frequency of action, but there is nothing characteristic.

Physical Signs.—The apex beat is displaced downwards and to the left. The visible pulsation is not necessarily extensive. Palpation reveals a slow and powerful impulse. Percussion may reveal an increase in the area of cardiac dulness. In hypertrophy of the left ventricle, the first sound is less distinct than normally, both at the apex and at the aortic region, whereas the second (aortic) sound is accentuated at both places. Reduplication of the first sound at the apex in hypertrophy of the left ventricle suggests that compensation is beginning to fail. Hypertrophy of the right ventricle or auricle is not recognisable by percussion unless dilatation is also present. Such enlargements will be referred to in connection with dilatation. Reduplication of the second pulmonic sound in hypertrophy of the right ventricle is attributable to the high tension in the pulmonary circulation, and accordingly suggests that the ventricle is still equal to its task.

In hypertrophy of the left ventricle the *pulse* represents the cause of the hypertrophy rather than the latter itself. It is therefore often a pulse of high tension, the wave rising gradually and the vessel being easily felt between the beats.

Prognosis.—This is really an estimate of the duration and completeness of compensation. If the cause of the hypertrophy is a stationary lesion, compensatory hypertrophy may be sufficient after some weeks or months, and may thereafter be satisfactory for an indefinite period. This may be the case in mitral regurgitation, and in aortic disease established in early life. If the cause is persistent or progressive—*e.g.*, high arterial tension or sclerosis of the aortic valve—hypertrophy may for a considerable time keep pace with the requirements, but in the long-run the myocardium tends to degenerate, compensation fails, and symptoms supervene. Failure of compensation may be precipitated by acute disease, insufficient food, or severe effort.

Dilatation.

Etiology.—Dilatation of a cardiac cavity is often beneficial, as in mitral regurgitation, where the left ventricle must be

able to accommodate the amount of blood which it forces into the arterial system, and in addition that which regurgitates. The same thing applies to aortic regurgitation. But in many cases the dilatation after a time is in excess of what is rendered necessary by the regurgitation ; and this excess is due to failing vigour of the myocardium. The muscle of the heart yields and becomes stretched under its burden, and the organ is thus unable to empty itself completely at the systole.

An undesirable kind of dilatation also occurs in the left ventricle, and usually at the same time in the right ventricle, apart from the valvular lesions mentioned. A common cause of this is high arterial tension, whether in Bright's disease or in the other circumstances which lead to arteriosclerosis. It is met with in chronic alcoholism, particularly in beer-drinkers. Any cause which leads to progressive hypertrophy of one or more of the chambers of the heart leads in the long-run, if not from the first, to dilatation also. The staying-power of the heart is less in some people than in others, and in such, especially if high tension is present, slight temporary causes which either augment the strain or diminish the strength may induce dilatation.

Dilatation, then, supervenes in the *left ventricle* in consequence of prolonged high tension, or habitual and excessive muscular exertion ; in aortic obstruction in the late stage, and in aortic regurgitation from the first. Dilatation also results from degenerative changes in the muscular fibres of the ventricle, as in anæmia, acute diseases, and disease of the coronary arteries.

Dilatation of the *right ventricle* results from mitral regurgitation, whether this be due to mitral endocarditis or to dilatation of the left ventricle, whose various causes have just been mentioned. The right ventricle also dilates in mitral stenosis ; in emphysema and other diseases of the lungs which raise the pressure in the pulmonary blood-vessels ; and in connection with pericarditis and adherent pericardium.

The *left auricle* undergoes great dilatation in mitral stenosis. The *right auricle* tends to undergo dilatation along with the right ventricle.

Morbid Anatomy.—Dilatation is generally accompanied by hypertrophy, the characters of which have been already described. The right ventricle suffers oftener than the left, for its wall is thinner, and the causes of dilatation are exceedingly common. In aortic incompetence with secondary incompetence of the auriculo-ventricular orifices, there may be great general enlargement of the heart, and, indeed, it is in this disease that the largest size of heart, the *cor bovinum* or bullock's heart, is observed.

Symptoms.—In the early stage, there may be general impairment of mental and bodily vigour, and perhaps fainting attacks and insomnia. Breathlessness is induced by slight exertion. Later on, there are dropsy beginning in the dependent parts and extending widely, coldness and lividity of the extremities, great dyspnœa or orthopnœa, and enlargement of the liver. The *irritable heart* of soldiers, characterised by palpitation and shortness of breath, represents an early stage of dilatation and hypertrophy from over-exertion.

Physical Signs.—The cardiac impulse is diffuse. It may be difficult to recognise any apex impulse, and what seems to be such may really be due to the right and not to the left ventricle. The area of dulness is increased horizontally. Murmurs are often present—indicative either of the valvular lesion that caused the dilatation, or of secondary incompetence resulting from the dilatation. Apart from murmur, however, the first sound in dilatation becomes short and sharp and sometimes unduly loud. Prolongation of the short pause at the expense of the long pause, so that it may be difficult to recognise which is the first sound, and a cantering rhythm of the heart's action, are other signs that may be present. The pulse is not characteristic; in advanced cases it is usually irregular, sudden, and easily compressed.

Dilatation of the *left ventricle* causes displacement of the apex beat outwards and downwards. Pulsation may be felt at the anterior or mid-axillary line, and in the sixth or seventh intercostal space. The area of dulness is increased in the same direction. Pulsation is diffuse, and a systolic

murmur may be heard at the apex in consequence of relative mitral incompetence.

Dilatation of the *right ventricle* causes diffuse heaving in the region of the ventricle and in the epigastrium. Pulsation in the epigastrium may be due to the ventricle directly, but is more likely to be communicated to the liver and so to the abdominal wall. The area of cardiac dulness is increased to the right and perhaps also to the left. A systolic murmur due to secondary incompetence of the tricuspid is often heard.

Dilatation of the *left auricle* is rarely capable of recognition by clinical methods. There is reason to believe that a presystolic pulsation which is occasionally observed to the left of the sternum in children with mitral stenosis may be due to a greatly enlarged left auricle.

Dilatation of the *right auricle* is scarcely to be separated from the similar and simultaneous change in the right ventricle. The area of dulness is extended to the right, and presystolic pulsation may possibly be detected to the right of the sternum.

Prognosis.—This depends partly on the degree of lesion as estimated by the symptoms and physical signs. The family history may throw much light on the capacity of the heart to recover. If any circumstance which has caused or aggravated the cardiac failure can be removed, the outlook is naturally so much the better. Much will be learned in a few days from the results of treatment.

Treatment.—Rest is essential. Mercurial purges should be given to lower arterial tension and to drain the portal system. In acute dyspnœa with lividity, venesection should be practised ; in less urgent cases, leeching may be employed. Dropsy should be got rid of by Southey's tubes or by the aspirator, and cardiac tonics, such as digitalis, strophanthus, strychnine, and ammonia, are indicated. In early stages, quiet and regular habits of life, light diet, and regulated exercise short of fatigue, are important.

In cases of dilatation following acute disease, the treatment employed by Schott of Nauheim is sometimes valuable. This consists of baths and exercises. The water is warm,

and contains the chlorides of sodium and calcium, together with free carbonic acid gas. The exercises are slow regulated movements of the limbs and trunk against gentle resistance. This treatment can be carried out at home. After a course of Schott's methods, the convalescent, if he is wealthy and wishes a change of scene, may employ Oertel's treatment. This is carried out at a height of some 2,000 feet above sea-level. The patient walks up a gentle slope each day, and the demands made upon the heart are gradually increased so as to promote hypertrophy. At the same time the diet is so arranged, and the fluids taken are so restricted, as to diminish the bulk of the circulating blood.

9. DISEASES OF THE MYOCARDIUM.

Etiology and Morbid Anatomy.—*Changes due to Disease of the Coronary Arteries.*—The main trunks of these arteries anastomose with one another, but their branches are usually end-arteries. The coronaries are very liable to atheroma, which narrows the lumen. A thrombus may form on the diseased patch, especially if the latter is calcified, and this may cause a complete block. Syphilis occasionally causes thickening of the arterial wall. Simple or septic embolism may obstruct small branches. The arteries are, moreover, liable to be obstructed at their orifices by atheroma of the aorta.

Sudden obstruction of a large branch causes anæmic necrosis or pale infarction (usually in the left ventricle), and sometimes *sudden death*. If the patient survives, the necrosed tissue undergoes softening (*myomalacia cordis*), and round-celled infiltration takes place at the periphery. The softened part may yield under the blood-pressure and give rise to *acute aneurysm*, and this in turn may lead to *rupture* of the heart. If the softened patch is small, the dead muscular tissue may be absorbed and replaced by fibrous tissue.

Slowly produced obstruction of one coronary may be

compensated for by dilatation of the other.[1] But slowly produced obstruction of a large branch, or sudden obstruction of a small twig, often leads to *fibrous transformation* (fibrous myocarditis, fibroid degeneration), one of the principal forms of interstitial myocarditis. The muscular fibres, starved of their blood-supply, undergo wasting, and are gradually replaced by fibrous tissue. To see this it may be necessary to slice the wall of the ventricle parallel to its surface. If an extensive area is thus affected, it may bulge and produce a *chronic aneurysm*. Obstruction of the coronary arteries, especially if slowly induced by atheroma of the aorta, may give rise to *fatty degeneration of the heart*.

Septic embolism may lead to abscess in the heart-wall, to aneurysm and rupture of the wall, and to pericarditis.

Atrophy.—In emaciating diseases, such as phthisis, the heart shares in the general wasting. The epicardial fat is diminished, and the muscle is dark in colour (brown atrophy) owing to increase or concentration of pigment-granules around the nuclei of the fibres.

Fatty Infiltration (*cor adiposum*, 'fatty growth on the heart') is sometimes an isolated condition, sometimes part of a general obesity. In either case there is a great excess of fat under the epicardium, and this may extend between the muscular fibres, sometimes penetrating the whole thickness of the myocardium. The vigour of such a heart must obviously be greatly impaired, but it is possible that the infiltration of fat is secondary to weakness and wasting of the muscle.

Fatty Degeneration occurs in anæmia, acute fevers, poisoning by phosphorus or arsenic, occasionally in alcoholism, and occasionally in consequence of obstruction of one or both coronary arteries. In well-marked cases, the inner aspect of the ventricular wall and the papillary muscles are streaked and spotted like a 'thrush's breast' or like a 'tabby

[1] It is not easy to accept as a literal statement of fact Clifford Allbutt's remark that 'the heart can hypertrophy, and in substantial measure of time and degree retain its hypertrophy, when its coronary arteries have both of them been very gradually but completely occluded' (*Lancet*, 1903, i., p. 649).

cat.' The degenerated part is soft and pale, and minute granules of fat are seen with the microscope inside the muscle fibres.

Parenchymatous Myocarditis (parenchymatous degeneration) is characterised by pallor and softness of the cardiac muscle. The muscle fibres contain non-fatty granules, and the striæ may be lost. The condition occurs chiefly in acute infective diseases such as pyæmia, diphtheria, and small-pox.

Interstitial Myocarditis occurs in rheumatism in connection with pericarditis and endocarditis. Syphilis may cause a localised interstitial lesion such as has been described in Section I. Fibrous tissue patches in the heart-wall are often the result of obstruction of a coronary artery.

Symptoms.—*Fibrous Transformation.*—The patient may die suddenly in the midst of apparently perfect health. Or there may be shortness of breath, palpitation and anginoid pains, perhaps induced at first by unusual exertion. The pulse is sometimes irregular, and may be either rapid or slow. The heart will probably be found enlarged, and there may be slight swelling of the feet. If the patient rests, recovery or great improvement may take place. Relapses, however, may be expected, and death may take place by gradual, rapid or sudden cardiac failure.

Fatty Infiltration may be associated with great shortness of breath, and may lead to sudden death, but it is not so serious a condition as fatty degeneration.

Fatty Degeneration is, like fibrous transformation, attended by the risk of angina pectoris and of sudden death. Those forms which accompany severe anæmias and acute fevers may be expected, and precautions ought to be taken to prevent exertion, such as sitting up in bed, which might overstrain the heart. The variety which develops in elderly people in consequence of obstruction of the coronary arteries cannot be anticipated in the same way, and in a great many cases the sufferer dies suddenly during or shortly after some mental or bodily exertion, or perhaps after a hearty meal which distends the stomach, without having previously complained of any symptom whatever. When symptoms do

occur, the early ones resemble those of dilatation as already described. The later ones, as Broadbent points out, are different, and are due to defective pressure in the arterial system ; they include syncopal, apoplectic, and epileptiform attacks, with the addition of angina pectoris, but without dropsy. Physical signs connected with the heart may be negative. The pulse may be very soft and very slow. Sudden death is often due to syncope, sometimes to rupture of the heart.

Stokes-Adams' disease or *syndrome* is a group of symptoms which includes slowness of the heart's action (usually permanent, but sometimes paroxysmal), and attacks of vertigo or syncope, sometimes with apoplectiform or epileptiform seizures. These apoplectiform attacks without paralysis have been called 'pseudo-apoplexy.'[1] Stokes-Adams' disease is met with in association with changes induced in the myocardium of young adults by syphilis, but it also occurs in older individuals who have arterio-sclerosis, and is probably due to a lesion in the medulla.

Prognosis. — This depends much on the cause of the disease in the myocardium ; when the latter is due to disease of the coronary arteries, the outlook is very grave, and there is considerable risk of sudden death. If the cause is removable, *e.g.*, acute fever or anæmia, complete recovery may be hoped for if suitable treatment is enforced.

Treatment.—When *disease of the myocardium* can be recognised during life, the patient must live a quiet life, with complete freedom from all mental and bodily strain, but with carefully regulated gentle exercise and perhaps massage. Strict moderation must be observed in food and drink, and the bowels must be kept regular. Any tendency to high tension should be counteracted by small doses of a mercurial laxative. In cases of *fatty infiltration* due to excessive eating and drinking, without fatty degeneration of the muscle, Oertel's treatment may prove of good service. This method includes restriction of the amount of liquid

[1] See Osler, 'Lectures on Angina Pectoris and Allied States' (1897), p. 70.

taken, a highly nitrogenous diet, and above all graduated exercises in hill-climbing at an altitude of about 2,000 feet.

Aneurysm and Rupture of the Heart.

Aneurysm.—Aneurysm of a valve has been mentioned in connection with endocarditis. Aneurysm of the heart-wall may be acute or chronic. The *acute* form may result from ulcerative endocarditis, acute myocarditis, or infarction from obstruction of the coronary artery. *Chronic* aneurysm is a result of fibrous transformation. In either case the softened and distensile piece of heart-wall yields under the pressure of the blood in the heart, and bulges outwards.

Aneurysm of the heart is much more common among males than among females. Acute inflammation acts as a cause chiefly in early life, degenerative change chiefly at and after middle life. The aneurysm is almost always connected with the left ventricle, and may be of any size up to that of a cocoanut. Muscular tissue may or may not persist in its wall. The pericardium over it is frequently adherent.

Any symptoms that may be produced by cardiac aneurysm are not characteristic, so that diagnosis during life is not practicable, and the aneurysm is only discovered, as a rule, after death. This may result from rupture into another chamber of the heart or into the pericardium, or from interference in some other way with the cardiac action.

Rupture of the Heart.—Among the causes of this rare event are acute softening from obstruction of a coronary artery, fatty degeneration, fatty infiltration, abscess, and gumma. The patient is generally in advanced life, and the heart is in a condition of fatty degeneration. The rupture most commonly involves the left ventricle,[1] and takes place during exertion. The patient, who has perhaps had no previous symptoms, may suddenly complain of intense suffering about his heart and die in a few seconds. In exceptional cases, however, life is prolonged for some hours or days, and recovery has actually been recorded.

[1] Richard Wagner died in 1883 from rupture of the *right* ventricle of a greatly dilated heart, which was also the seat of fatty degeneration. During the last few months of life there was much dyspnœa, especially after meals (Gould, ' Biographic Clinics,' 1904, ii., p. 116).

10. PERICARDITIS.

Etiology.—Pericarditis is very often due to the rheumatic infection, and is accordingly met with in some cases of chorea. The myocardium is generally, and the endocardium frequently, affected along with the pericardium. Pericarditis sometimes complicates pneumonia, either owing to direct extension of the inflammation, or through the organism reaching the membrane by the blood. It may be a sequel of scarlet fever. It may be tubercular, and then either an isolated lesion or part of a more general infection (see under Tuberculosis). It also occurs in Bright's disease, gout and scurvy. Suppurative pericarditis is met with in septicæmia and pyæmia, or as an extension from a neighbouring focus of suppuration, or as an isolated condition.

The common variety of pericarditis, such as is met with in rheumatism, is associated with a sero-fibrinous exudation. A hæmorrhagic inflammation may occur in malignant disease or scurvy. The tubercular form has been described at p. 132. Rheumatism and scarlet fever are specially related to the pericarditis of childhood ; Bright's disease and gout to that of later life. It is enough to allude here to the common *milk-spot* or *soldier's spot*, which is a chronic localised pericarditis, due apparently to the irritation caused by the heart beating against neighbouring structures ; it is sometimes recognisable during life by the scratching sound to which it gives rise.

Three important types may be recognised anatomically and clinically : (1) acute pericarditis with sero-fibrinous exudation, (2) suppurative pericarditis, and (3) chronic adhesive pericarditis or adherent pericardium.

Acute Pericarditis.

Morbid Anatomy.—The inflammation generally affects both visceral and parietal layers of the pericardium so that these become coated with fibrin. Owing to the rubbing of the lymph-coated surfaces, these present an appearance suggestive of pieces of bread-and-butter which have been put

together and then separated. In addition to the fibrin or lymph, serum is often present, sometimes in large quantity. It is usually reabsorbed. In slight cases the fibrin may also be absorbed, but more commonly it is gradually replaced by granulation tissue, and the two layers become united. Thus the pericardial sac is partially or completely obliterated, and the condition known as adherent pericardium is produced. Unless in slight cases, myocarditis is present along with the pericarditis ; there is round-celled infiltration of the interstitial tissue, with degenerative changes in the muscle fibres of the heart-wall, and the heart itself is dilated.

Symptoms.—In some cases, particularly in children, symptoms are few or almost awanting ; there may be no pain or discomfort, and it may be difficult to keep the patient in bed. As a rule, however, there is some suffering, and this may be intense. The symptoms include pain about the heart, or distributed more generally over the chest ; shortness of breath, sometimes amounting to orthopnœa ; pyrexia ; acceleration of the pulse ; restlessness ; insomnia ; and an anxious expression. In severe cases, there may be dysphagia owing to irritation of, or pressure on, the œsophagus where it passes close to the pericardium. Cough, from pressure on the trachea ; aphonia, from pressure on, or irritation of, the recurrent nerve ; vomiting ; and delirium with other head symptoms may also be present.

Physical Signs.—Where there is any considerable effusion, the cardiac dulness is increased at first upwards along the left border of the sternum. A large effusion causes the dulness to extend for some distance outside the left nipple line and to the right of the sternum. The apex impulse becomes feeble and may disappear. The heart's sounds are much weakened.

The most characteristic sign is the friction sound produced by the rubbing of the roughened surfaces. It is superficial and sometimes distinctly rubbing or scraping in character, and can be modified by the pressure of the stethoscope. It usually accompanies both the systole and the diastole, but may be systolic only. The friction sound has no particular seat of maximum intensity or direction of propagation like an endocardial murmur. It is often heard over the right

ventricle to the left of the sternum. It may shift its place from day to day, and may be modified by changes in posture. It is sometimes accompanied by friction fremitus. The friction sound tends to disappear owing to absorption of effused lymph, separation of the roughened surfaces by serous effusion, or permanent adhesion of these surfaces. As the effusion accumulates, the sounds of the heart may become very faint. As absorption takes place, the lost friction sound may return.

In adults the disease is generally acute. In the slightest cases there may be little or no increase of the cardiac dulness, and even in cases with distinct increase the height of the disease may be past in a few days, and convalescence will soon be complete—usually with adhesion of the two layers of the pericardial sac. In very severe cases death may take place in a few days, owing no doubt chiefly to the associated myocarditis and great dilatation of the heart ; for in such cases there may be very little liquid effusion.

In children, the onset is often insidious and the suffering slight, though there is likely to be pallor and shortness of breath. The disease is subacute in course, and often disables the heart permanently. Not uncommonly it is progressive or recurrent, and becomes associated with dropsy and other evidences of cardiac failure which lead up to a fatal issue. Considerable effusion may cause marked bulging of the præcordia, even in big children.

Diagnosis.—In connection with rheumatism and other known causes of pericarditis, the condition of the heart must be carefully watched. The unobtrusive character often presented by the manifestations of rheumatism in childhood must always be borne in mind. Pain about the heart, frequency of the pulse and shortness of breath should be carefully attended to. Friction sound is the most important sign ; it is not conducted in definite directions like endocardial murmurs, and it is often modified by pressure with the stethoscope. Pleuropericardial friction, due to roughening of the pleura where it overlaps the pericardium, is influenced by respiration, is heard not so much over the heart as at its borders, and is likely to be associated with pleural

friction elsewhere. The early enlargement of the cardiac dulness in an upward direction is an important sign of pericardial effusion as distinguished from enlargement of the heart.

Prognosis.—Acute pericarditis due to rheumatism is generally recovered from, at least for the time. In pneumonia it is a grave element in the case. In Bright's disease, the prognosis is unfavourable, largely on account of the depraved blood state. In subacute and recurrent cases, the outlook is grave, on account of the tendency towards progressive crippling of the myocardium. The chronic localised pericarditis which constitutes a 'soldier's spot,' and gives rise to a scratching sound during life, is of no importance clinically.

Treatment.—Absolute rest should be enjoined as early as possible, and any treatment indicated for the primary disease (*e.g.*, the salicylates in rheumatism) must be put in operation. Leeching and the ice-bag over the heart are indicated in the early stage. Blisters, diuretics and laxatives may be of service later on in promoting the removal of the effusion. If this is so large as to cause urgent symptoms, the sac should be aspirated, the puncture being made in the fifth left space, an inch or an inch and a half from the left sternal margin. The risk is diminished by making a preliminary incision down to the intercostal muscle, where possibly bulging may be detected. The right ventricle has been repeatedly punctured by mistake ; such an event may cause death or may do no harm.

Suppurative Pericarditis.

This disease is almost always purulent from the commencement.

Symptoms.—These are often very indefinite and of insidious onset. There is usually no pain, but the pulse is accelerated, and the breathing may be difficult. The temperature may be undisturbed or may suggest some septic condition.

Physical Signs.—There is increase of the cardiac dulness, but seldom any friction sound.

Diagnosis.—If the disease is suspected, the skin should be incised and an exploratory puncture made in the fifth left space, an inch or an inch and a half from the left border of the sternum. If pus is found, the treatment must be by incision and drainage.

Prognosis.—If the suppuration is local, there is hope of recovery. If it is part of a septicæmia or pyæmia, the general condition makes the case almost hopeless.

Adherent Pericardium (Chronic Adhesive Pericarditis).

Two varieties of this condition are recognised : (1) simple adhesion of the two layers of the pericardium to one another, in consequence of pericarditis ; and (2) adhesion of the two layers of the pericardium to one another, with adhesion of the parietal layer to the chest-wall and pleuræ as a consequence of chronic mediastinitis or mediastino-pericarditis.

(1) In the first case the obliteration of the cavity may be partial or complete. The heart is not necessarily disturbed, but it may be enlarged, as the myocardium is apt to be somewhat damaged. There are no distinctive signs or symptoms. Of course, valvular disease often coincides with adherent pericardium, and may lead to hypertrophy.

(2) The second variety is much more serious, especially in childhood, and may lead to great dilatation of the heart, such as may be recognised by inspection, percussion and palpation. The apex beat may be fixed, so that it does not shift when the posture is changed. The left costal cartilages and the lower end of the sternum may be dragged in by each systole ; recession of intercostal spaces is not so certain a sign, since it may be due to atmospheric pressure. Systolic retraction of the lower parts of the chest-wall at the back and left side, and a diastolic shock felt by the hand over the heart at the commencement of diastole, and due to the recoil of the chest-wall when the systole ceases, are two other signs that are described. The movement of the epigastrium in inspiration may be hindered by adhesions connecting the heart with the diaphragm.

The **symptoms** connected with this condition are those

of hypertrophy and dilatation of the heart, and ultimately of failure of the right ventricle. The diagnosis may be difficult, but if cardiac symptoms are present which cannot be explained by physical signs pointing to endocarditis, adherent pericardium should be thought of, and the various signs mentioned above should be looked for.

The **prognosis** in mediastino-pericarditis is not very favourable, especially if the heart is enlarged.

11. HYDROPERICARDIUM, HÆMOPERICARDIUM, AND PNEUMOPERICARDIUM.

Hydropericardium, or dropsy of the pericardium, may result from local pressure on veins, but is usually part of a more general dropsy, such as results from disease of the heart or kidneys. When the effusion is large, the cardiac dulness is increased and the sounds may be enfeebled, but the interpretation of these facts must often be doubtful, and the symptoms are usually lost in those of the cause of the effusion. If the condition could be recognised as causing cardiac embarrassment, paracentesis would be justifiable.

Hæmopericardium is to be distinguished from the hæmorrhagic form of pericarditis which may occur in scurvy and malignant disease, and sometimes in tuberculosis and Bright's disease. It may be either traumatic or idiopathic, the latter variety resulting from rupture of the heart, of a coronary artery, or of an aortic aneurysm. Death usually takes place quickly with symptoms of rapid failure of the heart. Occasionally after rupture of the heart the patient survives for some time, and there may then be physical signs of effusion, with symptoms of heart failure.

Pneumopericardium is very rare. It may be traumatic or may result from perforation of a pulmonary cavity, a gastric ulcer, cancer of the gullet, etc. Perforation is soon followed by acute pericarditis with purulent and perhaps sanguineous effusion.

The symptoms are to some extent those of acute pericarditis, but rigors and perspirations may be more marked. The gas in the pericardium yields a tympanitic note, and

the liquid naturally yields a dull note. The tympanitic area shifts about according to the posture of the patient. Moreover auscultation reveals curious splashing or churning sounds which result from the movements of the heart stirring up the mixture of air and liquid. Friction sound may be recognisable. Death usually takes place within a few days. Traumatic cases may recover, but cases resulting from perforation are almost hopeless, chiefly by reason of the primary disease.

In traumatic cases, operation would be justifiable.

12. NEUROSES OF THE HEART

Angina Pectoris (Stenocardia. Breast-pang).

This is a neurosis characterised in typical cases by attacks of intense pain in the region of the heart, with a sense of impending death.

Etiology.—The disease is most common in men, in advanced life and in the wealthier classes. In all these respects it resembles gout, with which moreover it is sometimes associated. Occasionally the tendency is inherited; thus Dr. Thomas Arnold of Rugby, his son Matthew, and his father all suffered from this disease and died suddenly.[1] It occasionally occurs in diabetic and in syphilitic subjects, and it may be a sequel of influenza. It sometimes, even in early life, attends lesions of the aortic valve, syphilitic aortitis, and adherent pericardium. It may be a complication of aortic aneurysm.

Morbid Anatomy.—In a large proportion of cases the heart is fatty, and when this is the case the coronary arteries are usually diseased, sometimes to an extreme degree. Occasionally there is fibrous transformation. Sometimes nothing abnormal is detected in the heart. The state of the heart in a paroxysm is unknown.

Symptoms in the Paroxysm.—The first attack almost invariably comes on during exertion, for instance walking quickly, or uphill, or against the wind. Mental excitement,

[1] Stanley's 'Life of Arnold,' ii. 282 *et seq.*; Osler, 'Angina Pectoris.'

exposure and indigestion are other causes. The onset is sudden, and the pain is an intolerable agony ; the patient feels that if it continues he will die. It is seated in the cardiac region or somewhere near there, but often radiates into the left arm, as far as the elbow or even the little finger. It may involve also the right arm and the left side of the neck, or some more distant part, and it may be accompanied by a desire to micturate. The sensory nerves from the heart are believed to enter the spinal cord by the eight or nine highest dorsal roots, and impulses originating in the heart will thus, by diffusing themselves in the grey matter of the cord, be reflected in part into the upper limb.

The expression is anxious, and pallor and sweating are often noticeable. The pulse may be small or unaffected. The attack often ends with an escape of wind from the stomach. It usually lasts a few minutes, but may be seconds or hours in duration. As the disease progresses, the tendency is for the attacks to be excited by less obvious causes and to be more severe in character.

In angina, however, the pain varies much in kind and in degree. In some cases, as was pointed out by Gairdner, there is no pain, but instead a strange, indescribable, subjective sensation. The condition may conform in other respects to angina pectoris, and was named by Gairdner *angina sine dolore*. In the *angina pectoris vasomotoria* of Nothnagel, there are coldness, pallor, palpitation, oppression, and a sense of impending death, but far less pain than in typical angina.

Angina pectoris often leads to sudden death, which may occur in a paroxysm or in an interval. The fatal paroxysm may be the first or a much later attack.

Pathology.—Various theories have been advanced as to the nature of this disease, as for instance that it is a neuralgia seated in the cardiac nerves, or that it is due to transient ischæmia of the heart in consequence of obstruction to the coronary circulation, or that it is due to overstrain of the ventricle. Allbutt holds that not the heart but the first part of the aorta is the seat of pain. Meanwhile as a provisional working hypothesis we may accept Broadbent's

view that 'stress is put upon the heart to which, for the moment, it is unequal.' In the majority of cases, there is persistent high arterial tension, and in many such cases the tension is further raised during the attack by contraction of the arterioles. In other cases, however, the tension seems to be habitually low, and arterial spasm will then add greatly to the strain upon a ventricle which may be degenerated. Broadbent thinks that the pain is actually defensive, in the sense that it compels the sufferer to stop all exertion at a moment when fatal arrest of the heart's action is threatened.

William Russell has recently advanced the theory that the attack of angina is due to general arterial contraction; and that this contraction may be the result of a normal reflex process associated with digestion, or may be caused by substances absorbed from the alimentary canal, or by emotion, or by effort. He further suggests that the vasomotor centre in the subjects of this disease has been rendered unduly sensitive by such agents as an excess of proteid food, alcohol, or tobacco; while, in addition, the heart may be embarrassed by the condition of the myocardium or of the coronary arteries.

Diagnosis.—Attacks of pain in the cardiac region are often complained of by persons who have not angina pectoris or any other kind of heart disease. Such attacks are sometimes spoken of as *pseudo-angina*. Except in connection with aortic valve disease or aneurysm, true angina is rarely met with in men under middle age, and seldom in women at any age. The fact that the earlier attacks are brought on by exertion is important evidence in favour of true angina. Pseudo-angina occurs at any age and is most common in women. The attacks come on spontaneously, and may last for an hour or more. Pseudo-angina is favoured by a neurotic tendency, and by the toxic conditions which result from abuse of tea or tobacco. Students and others who lean much against the table at which they are sitting may suffer from sudden pain in the cardiac region, which may, like angina, come on in the course of a walk, and may last for a few seconds.

Prognosis.—This is grave, but occasionally with care recovery may take place. When the attacks are brought on by exertion or by some other distinct cause, are associated with high arterial tension and are of short duration, the outlook is not quite so anxious, and the patient's condition is not so miserable as when the attacks occur without apparent cause, and in connection with low arterial tension ; or as when they occur by night, and persist for hours.

Treatment.—The patient should avoid mental excitement and severe muscular effort. He must exercise moderation in eating and drinking, and have his bowels carefully regulated. High tension should be treated by small doses of mercury. Tonics such as arsenic are indicated if the heart is feeble, and iodide of potassium is sometimes of service, possibly through some influence on degenerated arteries. The usual treatment for the paroxysm is the inhalation of nitrite of amyl. The patient carries in his pocket a supply of thin, silk-covered glass capsules, each of which contains 4 or 5 minims of the nitrite. Whenever the pain begins, he breaks one of these capsules between his finger and thumb and inhales the nitrite which escapes on to the cloth. Between the attacks, nitroglycerin is used in the form of the 1 per cent. official solution (liquor trinitrini). One minim diluted is taken three times a day to begin with, and the dose is gradually increased until, if possible, the pain is averted. Nitroglycerin in the form of the official tabellæ trinitrini sometimes gives more relief than nitrite of amyl. In cases where the nitrites fail, chloroform by inhalation, and morphine by hypodermic injection, should be employed to relieve intense suffering.

Besides angina pectoris, several other cardiac neuroses require consideration.

Palpitation has been already noticed as a cardiac symptom, but it may occur apart from organic heart disease. It consists in abnormally violent and rapid action of the heart, of which the patient is conscious. It is specially common in women, in emotional subjects, and in persons who by overindulgence in tea or tobacco reduce the tone of their nervous system. Hysteria, neurasthenia, indigestion,

anæmia, and sometimes acute fevers, are causes. This neurosis (as distinguished from palpitation in heart disease) is apt to set in, or to be most annoying, when the patient is resting. Apart from the violence, and usually also acceleration, of the heart's action, physical examination generally yields negative results.

Treatment.—In some cases, palpitation may be arrested by breathing deeply, slowly and regularly for a minute or two. A belladonna plaster should be applied over the heart, and above all the underlying condition must be remedied if possible. Treatment therefore will be directed to the stomach, nervous system, or other organs, as the case may require, and to the correction of wrong modes of living, eating and drinking.

TACHYCARDIA (*Rapid Heart*).—The frequency of the heart's action in this neurosis may be as great as three or four times the normal. It results from conditions similar to those which cause palpitation. It is most common in middle life, and a moderate degree of it may apparently be in some instances normal to the individual, since it may be discovered accidentally, and may be unassociated with other evidence of disease. In some cases tachycardia comes in paroxysms which last for perhaps an hour, and recur during many years. The sufferers may live to old age, but death sometimes occurs from heart failures. Tachycardia may, of course, be due to paralysis of the vagus nerve, as in multiple neuritis.

Treatment.—A quiet life, with plain fare, and full doses of bromide and belladonna are the remedies indicated. If there is considerable distress, the patient ought to rest in bed for several weeks or even months. Digitalis and potassium iodide may be tried in such cases.

BRADYCARDIA (*Slow Heart*) is occasionally a natural peculiarity of an individual.[1] It is not uncommon in patients who, after some febrile disease, or after parturition, are resting quietly in bed. It may also be present in fatty degeneration and fibrous transformation of the heart, and in injuries and diseases of the nervous system.

[1] Napoleon, who, it is to be noted, was an epileptic, had an almost constant pulse-rate of forty per minute.

Treatment is often unnecessary ; in the other cases it is that of the primary condition.

ARRHYTHMIA includes (1) intermittence and (2) distinct irregularity of force and rhythm. These have been alluded to as symptoms of heart disease, but they are common as neuroses.

(1) The intermittent pulse misses a beat from time to time. Usually, in such cases, the heart itself does not miss a beat, but gives one so small that the wave does not reach the wrist. The condition may be habitual or temporary. Habitual intermission may be unnoticed by the patient ; it has no serious significance and needs no treatment. Temporary intermission results from flatulence, tea-drinking tobacco-smoking, and various other causes ; it may occasion some discomfort to the patient, and should be treated by removal of its cause. As intermission may result from fatty degeneration, it is important to recognise this condition when present. A smart walk makes the pulse worse if the heart is degenerated, but improves it if the heart is sound.

(2) Irregularity is more serious than intermission ; like the latter, it may be habitual or temporary. The habitual form does not necessarily prevent long life and health. It is common in organic heart disease. The temporary form results from such causes as tea, tobacco, and flatulence, and these ought to be remedied.

13. DISEASES OF ARTERIES.

Hypertrophy.

In chronic Bright's disease, the muscular coat or media becomes increased in thickness to withstand the increased tension within the vessel. The radial artery becomes in this way firm and incompressible like a piece of cord. In the long-run, however, fibrous change may to a considerable extent take the place of muscular hypertrophy.

The expression 'hypertrophy' has also been applied to the enlargement of collateral vessels which takes place after obstruction of an artery.

Atheroma. Arterio-sclerosis.

These names, and also the expressions endarteritis deformans, endarteritis nodosa, and chronic endarteritis, are applied to a condition commonly met with in the arteries, and described by different writers in such varying language that much confusion exists. Atheroma or endarteritis deformans is the degenerative process which tends to involve the arteries in persons who have passed the prime of life. It may be regarded as the natural result of wear and tear in these vessels. Atheroma or endarteritis deformans is sometimes regarded as an advanced stage of arterio-sclerosis, but there is little doubt that the term arterio-sclerosis is frequently applied to the muscular hypertrophy and fibrous overgrowth of the media which result, as atheroma often does, from persistent high tension.

Etiology.—Atheroma (endarteritis deformans) is usually an indication of advancing years and tends to begin at forty or forty-five. But the longevity of the arteries varies immensely in different individuals, and peculiarities of this kind may run in families. Apart from the age of the vessels, the most important element in causing them to degenerate is the strain to which they are habitually subjected, so that persistent high tension is a most important cause of arterial disease. High tension may be the result of an inherited tendency, or of Bright's disease, lead-poisoning, gout, pregnancy, constipation, excesses in eating and drinking, or a laborious occupation. But there is good reason to believe that certain toxic agents, such as alcohol, syphilis, and lead, give rise to degenerative changes in the arteries through their direct toxic influence, and without necessarily causing increase of tension.

One effect of persistent high tension is hypertrophy of the muscular coats of the arteries with, of course, thickening of their walls. After a time, the tendency is for fibrous tissue to be added to, or replace the muscular tissue. This condition (specially striking in chronic interstitial nephritis) appears to be the arterio-sclerosis of some writers ; it will be alluded to again in connection with Bright's disease.

A second consequence of prolonged high tension is what is everywhere known as atheroma. The favourite seats of this disease are the aorta (chronic aortitis), the coronary arteries of the heart, and the cerebral arteries. This localisation is partly explained by the force of the blood-wave at the parts affected, a view which is corroborated by the occurrence of atheroma in the pulmonary artery when the right ventricle is hypertrophied. Syphilis may lead to atheroma by causing endarteritis of the vasa vasorum.

Morbid Anatomy.—The disease begins as a localised thickening of the intima underneath the endothelium. The atheromatous patches (endarteritis nodosa) are hard and white, and as they are due to connective-tissue proliferation, they really constitute at first a hypertrophy of the intima. But fatty degeneration ensues, beginning usually in the deeper part of the patch, and giving it a yellow colour. Either before or after fatty degeneration, the patch may become infiltrated with lime salts. When the fatty material accumulates, it constitutes an 'atheromatous abscess,' and if this ruptures into the lumen, it gives rise to an 'atheromatous ulcer.' Calcareous plates may also tear through the intima, after which a thrombus commonly forms at the site of rupture.

The condition of the media and adventitia varies ; they may be normal, or thinned, or inflamed, or the seat of fibrous change.

Results of Atheroma.—These include narrowing of the lumen of the artery, diminution of muscular contractility, rigidity, and weakening of the wall. Atheroma of the coronaries leads to serious effects upon the heart. Atheroma of the aorta may obstruct the orifice of a coronary artery. Atheroma of the cerebral arteries often leads to serious brain disease. Loss of contractility may be important in the case of the cerebral arteries. Rigidity is serious in the case of the aorta ; the vessel does not dilate properly with the systole of the heart, and accordingly does not recoil and force the blood onwards in diastole. Moreover the aortic arch is apt to be permanently dilated, or to become the seat

of aneurysm. Further, the loss of elasticity in the arterial system throws increased strain upon the left ventricle, which therefore becomes hypertrophied. But the increased force of the ventricular contraction acting upon diseased bloodvessels is apt to rupture the latter—an occurrence which is common in the brain ; or the heart may at last become unable to undergo any further hypertrophy, and so the symptoms of heart failure set in.

Symptoms and Physical Signs.—The radial pulse often presents the phenomena of high tension. Later on the difficulty of obliterating it by pressure—if the primary cause was high tension—will be partly due to thickening of the arterial wall. If arterial tension falls through failure of the heart, the thickness of the wall may still make the vessel incompressible. The left ventricle will be hypertrophied. As long as the tension remains high, the aortic second sound will be accentuated.

The phenomena mentioned thus far cannot be regarded as peculiar to arterio-sclerosis, since they are regularly present in chronic interstitial nephritis for example. Many of these patients die with cardiac symptoms, which result from failure of the enlarged ventricle, or from obstruction of the coronary arteries ; others with cerebral symptoms which result from rupture or obstruction of a diseased artery ; and others, again, with renal symptoms which result from concurrent interstitial nephritis.

Treatment.—This includes a careful regulation of the food and drink, both of which must be moderate in quantity and plain in quality. The bowels must be regulated, and a small dose of mercury should be given once or twice a week if the tension is high. The skin should be kept active by baths. Regular exercise, short of fatigue, should be enjoined. Alcohol should be avoided. Potassium iodide should have a trial. Cardiac, cerebral or renal symptoms will often require special treatment.

Fatty Degeneration.

Fatty degeneration occurs as a primary change in the intima, especially of the aorta. The affected part is yellow

and slightly raised. It is common in anæmia and debility, and is comparable to fatty degeneration of the cardiac muscle.

Calcareous Infiltration.

This infiltration takes place, not only in atheromatous patches, but also independently, though often in the same individual as atheroma, and it may be in a different part of the same artery. Chronic alcoholism is a cause of this condition. The calcareous deposit generally begins in the muscle-cells of the media, and involves such arteries as the femoral, brachial and smaller vessels. Rigidity of the radial artery is due to this cause oftener than to atheroma. Calcareous infiltration renders the vessels rigid, and, if extensive, leads to enlargement of the left ventricle. Such vessels may become dilated generally, or may be fractured and then become the seat of a local dilatation or aneurysm.

Endarteritis Obliterans.

This is a thickening of the intima due to proliferation of connective-tissue elements, without degeneration. Capillaries grow into the intima from the media. The condition is seen in the small vessels of organs which are the seat of chronic interstitial inflammation—*e.g.*, in granular kidneys. The lumen may be almost obliterated. The same designation is sometimes applied to syphilitic endarteritis.

Acute Aortitis.

Acute aortitis is a rare disease whose etiology is still obscure. It has been met with in association with acute specific fevers, pregnancy, and renal disease, as well as independently. The vessel is usually dilated, and its inner surface is rendered uneven by the presence of reddish, gelatinous-looking patches which are due to inflammation of the vessel-wall, and especially of the intima.

The symptoms include dyspnœa, acceleration of the pulse, and attacks of anginoid pain which are not related to exertion. There is no fever as a rule. There are no distinctive physical signs. The ordinary duration of the

disease is several months, and the patient usually dies in an anginoid attack. The treatment is similar to that recommended for angina pectoris.

Aneurysm.

Various kinds of aneurysm are recognised. Thus the *true* aneurysm is a localised bulging of a vessel in which the sac is still constituted by at least a portion of the normal vessel-wall. This aneurysm may be either *fusiform* or *sacculated*. A *false* aneurysm is one in which all the coats have ruptured. In the *dissecting* aneurysm, the inner part of the vessel-wall has ruptured so that blood is present among the layers of the middle coat.

Sometimes a direct communication takes place between an artery and a vein. The vein is then apt to become varicose, and the condition is called *aneurysmal varix*. Sometimes a sac is interposed between the artery and vein, and this constitutes a *varicose aneurysm*.

Many aneurysms come naturally under the consideration of the surgeon, but the most important and frequent belong to the province of the physician. Among them are those of the aorta, which will be considered in the present section ; those of the cerebral arteries, which are described in connection with diseases of the nervous system ; and those of the pulmonary artery, which are dealt with in connection with pulmonary tuberculosis at p. 120.

Aneurysm of the Thoracic Aorta.

Etiology.—The disease is one of adult life, and is much more common in men than in women, probably on account of the influence of occupation.

Atheroma is the most important local cause, and is almost always present. The disease of the intima damages the media, and thus allows bulging to commence. Two of the most common conditions that favour aneurysm probably operate by inducing atheroma ; first, a laborious occupation, by causing mechanical strain on the vessel, or by rupturing a damaged intima or media ; and, secondly, syphilis, by inducing endarteritis of the vasa vasorum. A history of

syphilis is obtained in about half the cases. Hereditary tendency, alcoholism, and the different causes of high tension and of atheroma will naturally favour the occurrence of aneurysm.

Morbid Anatomy.—The *fusiform* or cylindrical aneurysm is a general dilatation of the vessel for some part of its length. A dilatation of this kind, which is not uncommon in the case of the aortic arch, is lined throughout by the intima, but this is likely to be atheromatous, and the media may be greatly atrophied. The *sacculated* aneurysm bulges from only a part of the circumference of the vessel. The intima is incomplete, but can generally be traced for a little distance into the sac. The media is atrophied. The adventitia is the principal constituent of the sac-wall, and to this may be added inflammatory fibrous tissue, and contiguous structures. Moreover, as the sac grows, it becomes lined by successive layers of clot, and in exceptional cases these will completely fill the cavity. When this happens, the aneurysm may be regarded as cured.

Symptoms and Physical Signs.—These vary greatly according to the part of the vessel affected. Thus aneurysms of the ascending aorta and first part of the arch are termed by Broadbent 'aneurysms of physical signs,' whilst those of the transverse arch are called 'aneurysms of symptoms.' The ascending part of the vessel is superficial, freely movable, and not closely connected with important structures, so that an aneurysm readily becomes large and accessible to physical diagnosis before it causes pressure-symptoms. On the other hand, the transverse arch is deeply situated, and in intimate relation with nerves, vessels, trachea, gullet, etc., so that an aneurysm here is likely to cause pressure-symptoms before distinctive physical signs can be recognised. In a third class of cases, neither symptoms nor physical signs may suggest aneurysm. Thus, when the intrapericardial portion of the aorta is involved, death may occur from rupture before the condition is suspected.

Most of the phenomena of aneurysm may be arranged under two headings: physical signs and pressure effects;

but these are not mutually exclusive. The following are some of the *physical signs*.—Abnormal pulsation may be seen or felt, and this may be expansile if the sac-wall is thin, whereas it is likely to be a simple thrust if the sac contains much clot. Such pulsation is most commonly observed in the upper part of the chest to the right of the sternum. Sometimes the aneurysm forms a prominent tumour which

FIG. 28.—RIGHT RADIAL PULSE IN CASE OF SUSPECTED ANEURYSM. (Jas. M. L., æt. 35; cardiac pain, double aortic murmur, inequality of pulses, and inequality of pupils.)

may project through the sternum or costal cartilages. Sometimes a systolic thrill is felt, and there is often a palpable diastolic shock which results from the recoil of the sac-wall after distension; the latter, when present, is a most important sign. Percussion may be dull over the aneurysm. The second sound may be loud, ringing and of low pitch. It coincides in time and cause with the shock just mentioned,

FIG. 29.—LEFT RADIAL PULSE FROM SAME CASE AS FIG. 28, SHOWING MORE GRADUAL UPSTROKE AND DOWNSTROKE, ROUNDED SUMMIT, AND SMALLER VOLUME.

and the low pitch is due to the increased calibre of the vibrating aorta. A systolic murmur may be heard over some part of the aorta, and is of special value if no murmur is heard at the aortic cartilage. A single or double murmur heard at the aortic area is by itself of no value in the diagnosis of aneurysm. The tracheal breath-sounds may be well conducted to the manubrium by an aneurysm which is

in contact with both trachea and sternum. An aneurysm distal to the innominate artery, and proximal to or involving the left subclavian, often modifies the left radial pulse ; the wave of the latter is slow in rising, rounded at the top, slow

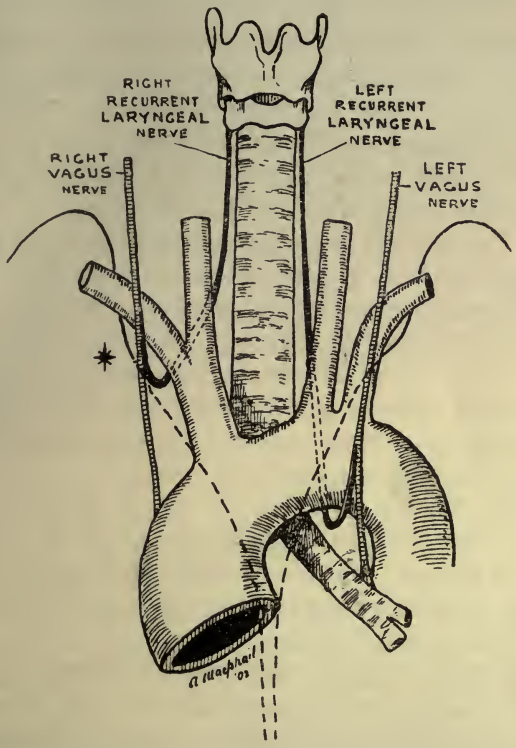

FIG. 30.—SKETCH SHOWING HOW THE LEFT RECURRENT NERVE IS LIABLE TO SUFFER IN ANEURYSM OF THE AORTIC ARCH, AND HOW THE RIGHT RECURRENT NERVE MAY SUFFER IN ANEURYSM OF THE INNOMINATE ARTERY.

The anterior margins of the pleuræ are indicated by interrupted lines below, and continuous lines above. The asterisk marks the place where the right recurrent nerve passes between the pleura and the commencement of the subclavian artery, and where it may be injured in connection with disease of the pleura, or by the pressure of enlarged lymph glands.

in subsiding, and altogether less ample, when compared with that of the right radial pulse (Figs. 28 and 29). The carotid pulses may differ for a similar reason. The heart may be displaced by a large aneurysm, but is not hypertrophied through this agency alone.

The following are the more important *pressure-effects*. Pain is the most common and sometimes the only symptom. It may be due to pressure on bones, nerves, etc., and is sometimes intense. It may be localised or may radiate widely. It may simulate angina pectoris. Inequality of the pupils is sometimes present, and is probably due to pressure on the sympathetic nerve of one side, though some recent writers have suggested that it is a result of post-syphilitic changes in the oculomotor nucleus. Pressure on the recurrent laryngeal nerve (generally the left) causes palsy of the vocal cord, with changes in the voice and cough. Pressure on the superior vena cava or left innominate vein may lead to enlargement of superficial veins and local œdema. Pressure on the trachea or bronchi may cause cough, stridor, and paroxysmal or continuous dyspnœa. Pressure on one bronchus causes enfeeblement of the respiratory murmur over the corresponding side of the chest. The tracheal tug is apt to be produced by an aneurysm of the inferior aspect of the aortic arch when it is so placed as to press upon the left bronchus or neighbouring part of the trachea. This downward tug accompanies the systolic distension of the sac, and is felt when the fingers are placed under the cricoid cartilage so as to pull it upwards. Pressure on the gullet causes dysphagia.

Aneurysm of the Intrapericardial Portion of the Aorta.—Symptoms and physical signs are often absent. Rupture usually takes place into the pericardium and causes sudden death. Sometimes, however, there is anginoid pain, or the superior vena cava is compressed.

Aneurysm of the Aorta between the Pericardium and the Innominate Artery is specially characterised by distinctive physical signs, but there may also be pain from erosion of the chest-wall, and evidence of pressure on the superior vena cava, or on the root of the right lung. The sac may rupture into the pleura, pericardium or superior vena cava.

Aneurysm of the Transverse Part of the Aortic Arch usually causes characteristic pressure-effects before the physical signs are distinctive. In addition, the radial

pulses are likely to be different. If the aneurysm grows to a large size, the various physical signs previously enumerated may be met with. The sac may rupture into the trachea, left bronchus, left pleura, or gullet ; or death may result from pressure-effects.

Aneurysm of the Descending Portion of the Arch. —Neither symptoms nor signs are very definite, and sometimes pain radiating from the back is the only evidence of the disease. There may, however, be compression of the gullet and left bronchus, and there may be dulness or even an external tumour in the scapular region. Rupture frequently takes place into the gullet or pleura.

Aneurysm of the Descending Aorta may erode the vertebræ, compress the spinal nerves, obstruct the gullet, and even give rise to a tumour in the back. Pain, however, may be the only symptom.

Diagnosis.—The principal condition that must be excluded is tumour of the mediastinum, in which case there is no expansile pulsation and no aneurysmal second sound of the heart. The history of the case may throw light upon it, and the X rays may show the presence or absence of expansile pulsation in a deeply-seated aneurysm or tumour.

Prognosis.—The patient is constantly in a state of danger owing to the possibility of rupture. Nevertheless, life is sometimes prolonged for many years, and occasionally healing takes place. Even though blood oozes from the aneurysmal sac into the trachea, the patient may survive for a long time. Intrapericardial aneurysms usually rupture early. Aneurysms of physical signs are less dangerous than those that compress important structures. Apart from hæmorrhage and pressure on the air-passages, death in aortic aneurysm is sometimes due to pulmonary tuberculosis, the result, it has been supposed, of pressure on the pulmonary artery.

Treatment.—The object is to get the sac filled with laminated clot, and this event is favoured by a slow circulation and a small volume of blood. Therefore the patient should remain in bed for six or eight weeks to begin with, and thereafter lead a very quiet life. The diet should not be

bulky, and the liquid should be restricted as much as possible. If Tufnell's treatment is to be strictly carried out, the patient should rest for several months without once sitting up even for a moment, and the ingesta should be restricted to 7 or 8 ounces of solids and the same quantity of milk. The bowels must be kept regular. The most important drug is iodide of potassium, which may be given in doses gradually increased from 10 up to 30 or 40 grains thrice daily. It is of value in relieving pain, and it is also supposed to favour coagulation in the sac. An opiate may be required for the relief of pain or restlessness. Venesection may be of great use, both for venous engorgement and for severe pain. In one case where the pain was chiefly troublesome at night, it was speedily removed by quinine, after other remedies, including venesection, had quite failed.

A method employed in recent years was the hypodermic injection of a 2 per cent. sterilised solution of gelatin. Three or four ounces of the solution might be injected into the abdominal wall every second day for several weeks. The gelatin was absorbed, and was supposed to increase the coagulability of the blood. A febrile attack, however, was apt to follow, and as the aim of treatment is to keep the circulation as calm as possible, this constituted a serious objection. Moreover, many cases of tetanus have followed injections of this kind, so that it is far better to give the gelatin solution by the rectum — a procedure which has been known to lead to complete consolidation of an aortic aneurysm.

Surgical measures are not to be recommended for aortic aneurysm, unless possibly when the orifice of the innominate, carotid or subclavian artery is involved, in which case the corresponding trunk or trunks might be ligatured distally to the aneurysm. Acupuncture, galvanopuncture, and the introduction of foreign bodies into the sac involve the risk of displacing clot, as well as the risk of hæmorrhage and fatal inflammation.

Abdominal Aneurysm.

The *abdominal aorta* is occasionally the seat of aneurysm, usually close below the diaphragm. Its etiology is similar

to that of thoracic aneurysm, but it is of far less common occurrence. It may be fusiform or sacculated. The sac may extend backwards, eroding the vertebræ, compressing the cord, and causing motor and sensory symptoms in the legs ; but more often it bulges forwards, and may then be recognisable as a definite tumour with expansile pulsation. A murmur may be audible over the sac, and there is severe, paroxysmal and radiating pain. Vomiting, attributable to irritation of the stomach, and modification of the femoral pulse may also result. The common neurotic pulsation or ' palpitation ' of the aorta must not be mistaken for the very uncommon aneurysm. The sac tends to rupture into the retroperitoneal tissues, peritoneum, intestine, pleura, etc. The treatment is similar to that of thoracic aneurysm, though, if the tumour is low down, prolonged compression of the vessel under chloroform may be tried.

The *cœliac axis*, or some other branch of the abdominal aorta, may be involved in aneurysm, either alone or together with the aorta.

Arterio-venous Aneurysm.

This used to be common at the elbow when venesection was much practised. It is occasionally produced by rupture of an aneurysm of the ascending aorta into the superior vena cava, an occurrence indicated by the following signs : (1) cyanosis, œdema, and venous engorgement in the territory drained by the superior cava ; (2) sudden onset of symptoms ; (3) physical signs of aortic aneurysm ; and (4) a continuous murmur intensified at each systole.

SECTION IV

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

DISEASES OF THE BLOOD

i. The Blood.

THE most important points to be borne in mind in connection with these diseases concern the red corpuscles (*erythrocytes*), the white corpuscles (*leucocytes*), and the hæmoglobin.

The red corpuscles are yellow biconcave discs, 7 μ in diameter, which are derived, in the adult, from the nucleated red corpuscles in the bone-marrow. They are present in the blood to the number of about 5,000,000 per cubic millimetre in men, and about 4,500,000 in women. Their number is reduced (*oligocythæmia*) in anæmia, and increased (*polycythæmia*) in cyanosis. They may vary also in shape and size. Abnormally large red corpuscles (*megalocytes*) are common in pernicious anæmia. Small ones (*microcytes*) occur in this and other kinds of anæmia. Abnormally shaped corpuscles (*poikilocytes*) occur in any severe anæmia. Under the same circumstances the staining reaction of hæmoglobin may be lost; instead of staining pink with hæmatoxylin and eosin, the corpuscle may stain of a bluish tint (*polychromatosis*). Another abnormality is the presence of nucleated red corpuscles in the circulating blood; in health such cells are present only in the bone-marrow. Of the four varieties of nucleated red corpuscles—*normoblasts*, *megaloblasts*, *microblasts* and *poikiloblasts*—the two first are most important. Megaloblasts are almost confined to pernicious

anæmia, but are also observed in the curable anæmia due to *Bothriocephalus latus*. Normoblasts are present in splenomedullary leucocythæmia, and to a less extent in other forms of anæmia. Microblasts are sometimes observed in pernicious anæmia.

The hæmoglobin of the red corpuscles is the oxygen carrier, and hence, in order that respiration may go on properly in the tissues, it is necessary not only (1) that the lungs should be healthy, but also (2) that the heart should be healthy, and (3) that the hæmoglobin should be sufficient ; if any one of these three requirements fail, shortness of breath readily sets in. Defects in the number of red corpuscles and in the hæmoglobin do not necessarily keep pace with one another. The richness in hæmoglobin of the individual corpuscle is called the *colour index*, the normal being regarded as unity. By certain instruments which are in constant use clinically, we can determine (*a*) the number of red corpuscles in 1 cubic millimetre of blood. As the normal is 5,000,000 in men, and 4,500,000 in women, we can deduce the percentage richness of the particular specimen of blood in corpuscles. We can also determine (*b*) the percentage richness of the blood in hæmoglobin. The hæmoglobin percentage divided by the corpuscle percentage is the colour index, which in normal blood is equal to unity. In chlorosis the hæmoglobin defect is much greater than the corpuscular defect, and thus the colour index is less than 1. In pernicious anæmia, on the other hand, the colour index is greater than 1. In secondary anæmias the colour index is slightly below the normal.

White corpuscles or leucocytes are classified in various ways by different writers. The most common variety is the *polynuclear leucocyte* (polymorphonuclear neutrophile, finely granular oxyphile). Its nucleus often seems to consist of several nuclei partly fused together, or to be shaped like the letter S or Z. Cells of this kind constitute about 70 per cent. of the leucocytes of normal blood, and are also the common pus corpuscles. Another leucocyte (4 per cent. of leucocytes in blood) is the *cosinophile* or coarsely granular oxyphile ; its granules are large, and stain strongly with

eosin. Two other types are the *lymphocyte* and the *large mononuclear leucocyte* (though the latter is regarded by some as a large lymphocyte). These contain no granules, and are hence termed 'hyaline.' They possess a single nucleus. The lymphocytes amount to 20 or 25 per cent., and the large hyaline cells to about 5 per cent. of the leucocytes of the blood. Yet another variety is the *mast-cell*, which contains basophile granules. In medullary leucocythæmia and some other diseases *myelocytes* appear in the blood. These are large granular cells, which are normally present in bone-marrow. Their two varieties, neutrophile and eosinophile, are the mother-cells, in normal marrow, of the polynuclear neutrophile leucocyte and the eosinophile leucocyte respectively.

There are usually 7,000 or 8,000 leucocytes in a cubic millimetre of blood, but an increase in the number (*leucocytosis*) may occur under normal as well as under pathological conditions. Thus, leucocytosis is present during the digestion of food, and in connection with inflammations. It is the rule in some diseases, such as pneumonia, and the exception in others, such as enteric, and hence may be of diagnostic value. *Leucopenia*, or diminution of leucocytes, is not so common as leucocytosis. It is seen in the late stages of enteric fever, in pernicious anæmia, and in some other anæmias. Leucopenia in a well-marked case of pneumonia is rather ominous.

ii. Anæmia.

Anæmia is a general term used when the blood is defective in quantity, or in quality, or in both respects. Such defects may be either local or general, but it is only the latter that have to be considered at present. Hæmorrhage naturally causes a quantitative defect (*oligæmia*), but the quantity of the blood is quickly made up by the absorption of liquid from the lymph spaces and alimentary canal, so that the defect becomes a qualitative one. The most common anæmias are characterised by relative scantiness of the red corpuscles *oligocythæmia*), or of the hæmoglobin (*oligochromæmia*), or

of both. In certain other anæmias, the most striking changes are in the white corpuscles.

From the clinical point of view, anæmia is regarded as being either primary or secondary (symptomatic). Primary anæmia arises without discoverable cause, and is hence referred to a supposed disorder of the blood-forming or blood-destroying mechanism. Secondary or symptomatic anæmia, on the other hand, can be accounted for by the existence of a recognised cause, such as want of food ; or some digestive disorder which prevents the formation of blood ; or a toxic condition which causes destruction of blood ; or albuminuria, suppuration, or hæmorrhage, which impoverishes the blood in some of its constituents.

iii. Anæmia after Hæmorrhage.

Here the watery and saline constituents of the blood are quickly replaced. Some weeks elapse before the red corpuscles are present in normal number, while the restoration of the hæmoglobin takes several months. During the period of recovery, normoblasts and poikilocytes may be observed, and there is usually leucocytosis. The regeneration of blood depends upon the red marrow of bone, and this may be hypertrophied so as to replace much or all of the fatty marrow of long bones.

The hæmorrhage may be traumatic or spontaneous ; a single severe bleeding (*e.g.*, from the uterus), or repeated smaller ones (*e.g.*, from piles or a gastric ulcer). The symptoms include pallor, mental and bodily weakness, giddiness, shortness of breath, a soft rapid pulse, dimness of vision, ringing in the ears and faintness.

The treatment includes arrest of the hæmorrhage and removal of its cause, where this is possible ; rest in bed, abundant light food, and iron and arsenic internally.

iv. Chlorosis (Green Sickness).

Definition.—An anæmia of frequent occurrence in adolescent females, and characterised especially by deficiency of hæmoglobin.

Etiology.—The immediate cause is unknown. The disease is almost confined to females, and usually begins at puberty or within the next few years, though recurrences often take place later. Virchow's theory that it depends on imperfect development of the arterial system, and Sir Andrew Clark's view that it is the result of constipation and intestinal absorption, are both insufficient. There is no doubt that defects in the quantity and quality of the food, sedentary occupations, and long hours in ill-ventilated and badly lighted rooms are contributory causes.

Symptoms.—The face has often a greenish-yellow colour from which the name of the disease is derived. The cheeks may be ruddy, or may be pale and flush too readily. The subcutaneous tissue is well preserved or increased. Among the common symptoms are shortness of breath, palpitation, a tendency to faint, slight swelling of the ankles, gastric disturbance, constipation, and menstrual disturbance.

The *blood* is pale. The red corpuscles are generally less abundant than normally, but the hæmoglobin is much more reduced. On the average the corpuscles are about 80 per cent. and the hæmoglobin 40 per cent. of the normal, so that the colour index is $\frac{1}{2}$. The microscope shows pallor of the corpuscles, and in severe cases poikilocytes, normoblasts, and defective rouleau-formation. The leucocytes are normal.

A systolic murmur is commonly heard at the pulmonic area. A feature of this murmur is that it is less marked in the erect than in the recumbent posture. It is described as 'hæmic' or 'anæmic' because it depends on the condition of the blood. In well-marked cases, the murmur is heard all over the cardiac area, though loudest at the second left space. The murmur as heard at the apex is attributable to relative incompetence of the mitral valve. Under such circumstances, the apex beat may be found to be slightly displaced towards the left, while the area of dulness is increased. Though it might not be expected *a priori*, the pulse tension may be rather above the normal in the early stages of chlorosis, a condition which is explained by the capillaries and arterioles offering greater resistance to the flow of blood of abnormal quality than to that of healthy

blood. In time, however, the impure blood leads to degeneration of the cardiac muscle, which then yields under the strain; the ventricle thus dilates, the tension falls, and the mitral valve may also become relatively incompetent.

The mechanism of the hæmic murmur heard over the pulmonary artery is not fully understood. It has been explained as being really a murmur of mitral regurgitation conducted to the pulmonic area by the left auricle, but this view does not satisfactorily accord with facts.

A continuous murmur (the venous hum, humming-top murmur, or *bruit de diable*) is sometimes heard when the stethoscope is placed over the jugular vein, and is doubtless largely due to pressure by the stethoscope.

Complications.—Venous thrombosis is an occasional complication of chlorosis, and affects chiefly the veins of the lower limbs and the cerebral sinuses. Optic neuritis occurs in rare instances. Dyspepsia is common, and constipation is the rule. Superacidity of the gastric juice is also common, and gastric ulcer is a frequent complication. Menstruation is often absent, frequently scanty or irregular, occasionally excessive. Headache and neuralgia are common.

Diagnosis.—This depends on the sex and age of the patient, the peculiar colour of the complexion, the good general nutrition, the menstrual disturbance, the hæmic murmur, the characters of the blood, and the absence of any apparent cause of anæmia.

Prognosis.—The disease almost always proves curable under suitable treatment. Relapses, however, may occur, and there is the risk of thrombosis in the cerebral sinuses, the risk of embolism from a thrombus elsewhere, and the very faint risk of optic neuritis. A few cases obstinately resist treatment.

Treatment.—Unless the case is very slight, the patient should rest in bed. She ought to have abundance of fresh air and sunlight. The bowels must be kept regular, and for this purpose cascara sagrada or aloes may be given each night in a dose just sufficient to procure one evacuation the following day. Sometimes an alkaline bitter tonic may be

desirable. The specific remedy for chlorosis is iron, and this is conveniently given in the form of Blaud's pills. At first one pill of 5 grains may be given thrice daily after meals; but the dose should be increased once or twice a week by one pill, till after a few weeks the patient is taking six pills three times a day. After she is cured, she should continue to take iron in smaller doses for several months. The saccharated carbonate is another excellent preparation of iron; or the tincture of the perchloride may be employed. In cases where shortness of breath is a prominent symptom, arsenic should be given as well as iron.

v. Pernicious Anæmia

(Idiopathic Anæmia. Essential Anæmia. Progressive Pernicious Anæmia).

Definition.—An anæmia characterised by great reduction in the number of red corpuscles, and almost always progressing, though often with temporary remissions, to a fatal termination.[1]

Etiology.—Opinions differ as to whether this is a primary or a secondary anæmia. It is now well known that the clinical features of pernicious anæmia may be exactly simulated by certain anæmias which are undoubtedly secondary; *e.g.*, those due to cancer of the stomach, to *Bothriocephalus latus* or to *Ankylostoma duodenale*. But while some would therefore assert that the disease is always secondary, it is still true that in many cases no primary lesion can be detected. Stockman suggests that anæmia of a more ordinary kind may be changed into pernicious anæmia through the occurrence of multiple small hæmorrhages. Some cases follow parturition.

The defect of the red corpuscles must be due to diminished production or increased destruction. The latter is the more

[1] The first complete description of the disease was given by Addison, who called it idiopathic anæmia. It was while trying to find out the cause of this disease that he discovered the relationship between changes in the suprarenal capsules and the peculiar symptoms of the malady that has since been known by his name. His account of the two affections was published in 1855.

recent view, and has been ably advocated by Dr. William Hunter. He holds that pernicious anæmia is a chronic infective disease of the alimentary mucous membrane. He believes that the organisms of this infection produce toxins which destroy the red corpuscles in the portal system.

Pernicious anæmia generally occurs after full maturity is attained, and affects the two sexes almost equally.

Morbid Anatomy.—The skin has a lemon-yellow colour. The body is not emaciated. The heart muscle is in a state of intense fatty degeneration, so that the inner surface of the left ventricle has the colour of a thrush's breast. Fatty degeneration of bloodvessels and hæmorrhages are common. The yellow marrow of long bones is replaced by a red lymphoid tissue which often contains many megaloblasts. This represents a compensatory hypertrophy of the red marrow, but the numerous megaloblasts or gigantoblasts found in the hypertrophied red marrow of pernicious anæmia distinguish it from that of other anæmias, where the hypertrophy is normoblastic in type. The megaloblastic is imperfect as compared with the normoblastic type, since when these cells are liberated into the blood as oxygen-carrying red corpuscles, the large ones possess a relatively smaller respiratory surface than those of normal size. Atrophy of the gastro-intestinal mucous membrane and sclerosis of the spinal cord are occasionally found. It is probable that both these changes, as well as the megaloblastic degeneration of the bone marrow, are due to the same cause as the anæmia, viz., to the influence of some toxin.

One of the most constant and characteristic changes—one which is not found in the anæmias of malignant disease and hæmorrhage—is a great excess of iron-containing pigment (hæmosiderin) in the liver and kidneys, but not in the spleen.[1] This pigment is mostly in the outer zones of the hepatic lobules, and is recognised by a blue coloration on the addition of dilute (2 per cent.) hydrochloric acid and solution of ferrocyanide of potassium. It is supposed to be derived from the hæmoglobin of the corpuscles destroyed. The pigment

[1] Some writers speak of an increase of iron in the spleen, but this is certainly not always the case. See, further, William Hunter, 'Pernicious Anæmia' (1901), pp. 89-91, and Chapter IX.

is in the form of yellow granules, in which the iron is present in loose combination.[1]

Symptoms.—The onset is insidious. Languor, weakness, and shortness of breath are prominent symptoms from first to last. The pallor of the skin has often a lemon-yellow tint. The subcutaneous fat is well preserved. There may be slight œdema of the ankles.

The fresh blood looks watery. The red corpuscles are greatly reduced in number, and are often below 1,000,000 per cubic millimetre ; from 1,000,000 to 1,500,000 is a common figure. The hæmoglobin is also reduced, but seldom to the same extent as the red corpuscles, so that the colour index is above unity—perhaps 1·3. Sometimes it is more than 2, but occasionally it is less than 1. This relative richness in hæmoglobin of the red corpuscles is, with the doubtful exception of the prevalence of megaloblasts, the most important change in the blood, though it is not constant. The red corpuscles vary much in shape and size ; some are larger (macrocytes) and others smaller (microcytes) than normally. Some are irregular (poikilocytes), and some are nucleated (erythroblasts). Of erythroblasts, megaloblasts are more numerous than normoblasts. This condition of blood is not absolutely characteristic of idiopathic anæmia, though very suggestive.

Blood crises are occasionally observed, and are characterised by a great temporary increase in the number of nucleated red corpuscles. These crises may be either megaloblastic or normoblastic.

The white corpuscles are fewer than normally (leucopenia). While the polynuclear neutrophiles are diminished, the lymphocytes are increased relatively and sometimes absolutely. A few myelocytes are frequently to be found.

The pulse is accelerated and soft, and the murmurs of anæmia are heard over the cardiac area. Hæmorrhages are common, especially in the retinæ, but sometimes from the nose, uterus, and other parts. Vomiting and other disturb-

[1] The iron which is bound up in firm combination in the hæmoglobin molecule is not revealed by this test, so that the value of the latter is in no way influenced by the amount of blood present in the tissue examined.

ances of the digestive system are frequent. Sometimes the urine is deeply coloured from excess of urobilin. Slight pyrexia is present in many cases, either continuously or from time to time. The sternum and other bones may be tender. Death usually results from the anæmia itself, the patient being worn out by the headache, vomiting, palpitation and air-hunger.

Diagnosis.—The patient is usually of middle age. The symptoms set in gradually without discoverable cause, and run a progressive course. The characters of the blood, the irregular pyrexia, the lemon-yellow skin, the disorders of the alimentary tract, the retinal hæmorrhages, and the absence of emaciation are collectively almost pathognomonic. The stools should be examined to exclude intestinal parasites, and special care must be taken to exclude cancer of the stomach. With regard to the blood, the most important points are the marked oligocythæmia, the high colour index, the leucopenia, and the erythroblasts, most of which are megaloblasts. Apart from pernicious anæmia, a megaloblastic type of blood is seen in only two conditions, viz., (1) the secondary anæmia due to certain intestinal parasites, and (2) nitrobenzol poisoning. It is to be noted, however, that the blood may not at a given time present all the typical changes. As a result of treatment, or during a temporary remission, one or more of these may be absent. For instance, the colour index may fall below unity, and the anæmia thus assumes for the time the chlorotic type. In ordinary circumstances, pernicious anæmia can be diagnosed from the blood alone.

Prognosis.—This is always grave. Death generally occurs within a year, and often much earlier. Sometimes life is prolonged for several years, and temporary improvement is not uncommon under treatment. A few cases recover completely, but usually, it is to be feared, only temporarily. In one of Bramwell's cases, twelve years of good health intervened between the original attack and the fatal relapse.

Treatment.—Rest in bed is essential, and the diet should be light and nourishing. Iron is generally useless and is sometimes hurtful, but it should have a trial in cases where the colour index is less than unity. Arsenic is by far the

most valuable remedy, and undoubtedly prolongs life in this disease. Indeed, the immediate prognosis seems to depend on the patient's ability to take large quantities of arsenic, more than on any other factor. Fowler's solution should be given in 3-minim doses thrice daily, and the dose should be rapidly increased to 10 or more minims. Good results have sometimes been obtained by the use of intestinal antiseptics (salol, β-naphthol, bismuth salicylate), and in every case any existing catarrh or septic condition of the mouth or other part of the digestive tract should be promptly rectified. Bone-marrow, inhalations of oxygen and transfusion of defibrinated human blood have been recommended, and may be tried, but are quite unreliable. Hunter has reported a case of complete recovery under treatment by oral and gastric antisepsis (carbolic acid solution for the mouth, and bichloride of mercury for the stomach), with injections of antistreptococcic serum, followed by the administration of Fowler's solution. In a recent case which recovered in my wards the treatment included thorough cleansing of the teeth with carbolic solution, the administration of arsenic and bismuth salicylate internally, and injections of antistreptococcic serum hypodermically.

vi. Leucocythæmia (Leuchæmia).

Definition.—A disease characterised by a persistent increase in the number of white corpuscles in the blood, and by changes in the spleen, bone-marrow and lymphatic glands, either separately or together.[1]

There are two well-recognised types: (1) In medullary (spleno-medullary or lieno-myelogenous) leucocythæmia, the changes are principally in the spleen and bone-marrow. The increase of white corpuscles in the blood is due to cells derived from the marrow, viz., granular cells (myelæmia), and it has been supposed that the spleen is but a passive agent, or a storehouse for the cells (myelogenous leucocythæmia). Muir, however, has suggested that as one of the functions of the spleen is to remove abnormal elements from

[1] First fully described in 1845 by Hughes Bennett and Virchow independently.

the blood, its enlargement in leucocythæmia may be due to an effort to remove the excess of leucocytes from the blood. Why the marrow should become so active is quite unknown. (2) In lymphatic leucocythæmia, which is much less common, there is enlargement of lymphatic glands, and the increase of white corpuscles in the blood is due to lymphocytes (lymphæmia).

Etiology.—The disease is most common in middle life. Males suffer more than females. There is frequently a history of malarial infection. The immediate cause of the affection is unknown, though tumour-formation and infection by a protozoon or some other agent have been suggested.

Morbid Anatomy.—The blood after death has an almost pus-like appearance, and the clots found in the heart have a yellowish-green colour.

(1) In the medullary form, the spleen is greatly enlarged, and may weigh many pounds. The capsule is thick, and adherent to neighbouring structures. The enlargement is due to distension of the reticulum of the pulp with leucocytes like those in the blood. The fatty marrow of the bones is replaced by a tissue akin to red marrow. This tissue is sometimes like pus, but more often firm and pink. Even the bone may be atrophied by the growing marrow. This altered marrow contains myelocytes, nucleated red corpuscles, and polynuclear leucocytes. The change in the marrow is to be regarded as a hyperplasia, though later on there may be degeneration. In the lymphatic glands, and in the connective tissue of the portal area in the liver, there may be some little accumulation of leucocytes like those in the blood; but in this type of leucocythæmia there is not much tendency towards a general involvement of the tissues, just as in health the granular leucocytes are almost confined to the blood and marrow. Hæmorrhages may be found in the retina, skin, brain, etc.

(2) In the lymphatic form, there is general enlargement of the lymphatic glands throughout the body, the glands being packed with lymphocytes like those in the blood. In chronic cases the spleen may be greatly enlarged through distension of the pulp with lymphocytes; whereas in acute cases it may be scarcely enlarged at all. As a rule, the

Malpighian bodies are not affected. The bone-marrow is altered, and to the naked eye is often very similar to that of medullary leucocythæmia ; but the microscope shows that the change is due to infiltration with lymphocytes. The lymphoid tissue of the tonsils, pharynx, and intestine may be increased in bulk, the liver may be enlarged, and even such parts as the lower eyelid may be swollen and firm—in every case by infiltration with lymphocytes—while the connective tissue remains passive.[1]

Symptoms.—The symptoms are closely alike in the two forms of the disease. The onset is insidious, with increasing weakness and enlargement of the lymphatic glands or of the abdomen. Gastro-intestinal disturbance, epistaxis and menorrhagia are common early symptoms, and as the disease progresses, dyspnœa, emaciation and perhaps dropsy are observed. Hæmorrhages are common, and, if occurring in the stomach or in the brain, may cause death. The retinæ may be the seat of hæmorrhagic inflammation or of leucocyte infiltration. Priapism is sometimes a symptom, and is probably due to thrombosis in the corpora cavernosa. Examination will of course reveal the enlargement of the spleen or of the accessible lymph glands, and there may also be enlargement of the liver. In one case where the patient improved and was able to go about for a long time the spleen became dislocated to the lower part of the abdomen on the right side, and caused a bulging of the roof of the vagina to the right of the cervix. There may be pressure symptoms suggesting enlargement of the bronchial glands ; and possibly tenderness of the sternum in connection with changes in the bone-marrow. Moderate fever is present in many cases, often, however, alternating with periods of apyrexia. The excretion of uric acid in the urine is increased. The sputum may contain eosinophiles and both kinds of myelocytes.

The changes in the blood constitute the most important means of diagnosis. (1) In medullary leucocythæmia, the white corpuscles number perhaps 300,000 or 500,000 per cubic millimetre (instead of the normal 8,000), and are to the red corpuscles in the proportion of 1 to 10 or 1 to 5 (instead

[1] R. Muir, 'On Leucocythæmia, Lymphadenoma, and Allied Diseases' (*Glasg. Med. Jour.*, September, 1905, pp. 161-174).

of 1 to 600). As a rule, the polynuclear neutrophiles are most abundant, but many of the leucocytes are neutrophile myelocytes. (The latter are the mother-cells of the former, but under normal circumstances are not present in the blood of adults, though present in the bone-marrow.) Eosinophile leucocytes are absolutely, and in some cases relatively, increased; and many eosinophile myelocytes (mother-cells of the eosinophile leucocytes, and not normally found in the blood) may also be present. Basophile cells (mast-cells) may be few or many. Since the increase is in the granular cells, the lymphocytes are of course relatively diminished. The red corpuscles and hæmoglobin are diminished, and normoblasts are common. The colourless octahedral Charcot-Leyden crystals are found in the blood after death, and also on blood-slides which have stood for a time. (2) In lymphatic leucocythæmia, the increase of leucocytes is not nearly so great as in the other form. They number perhaps 140,000 per cubic millimetre, with a ratio to the reds of 1 to 20. The increase is only in lymphocytes, which amount to over 90 per cent. (instead of about 25 per cent.) of the leucocytes. The predominating type of lymphocyte may be large (especially in acute cases), or small (especially in chronic cases), or there may be a considerable variety of size. The granular cells are proportionately, if not absolutely, diminished. Myelocytes are not present as a rule. The red corpuscles and hæmoglobin are diminished.

Diagnosis.—This depends on the course of the symptoms, the enlargement of spleen or lymph glands, and above all on the characters of the blood.

Leucocythæmia must be distinguished from the *leucocytosis* of health and of inflammatory and other morbid conditions. In leucocytosis there are no myelocytes, and there is not a preponderance of lymphocytes. The enlarged spleen of leucocythæmia, extending downwards from under cover of the left lower costal margin, with its firm, smooth surface and rounded, notched edge, ought not to be mistaken.

Prognosis.—Death generally occurs within three years. It may result from hæmorrhage, or may be due to diarrhœa, dropsy and emaciation. The opening of a large abscess in such patients involves the risk of fatal hæmorrhage. Cases

sometimes undergo temporary improvement, and recovery has occasionally taken place.

Treatment.—The patient should have rest, fresh air and good food. Arsenic is the most important drug, and should be given in doses of 5 to 15 minims of Fowler's solution thrice daily. Quinine should be given in malarial cases. The combination of oxygen inhalations (30 to 60 litres, or 1 to 2 cubic feet daily) with arsenic internally sometimes gives good results. Exposure of the patient to the X rays is said to have done good in some instances. Splenectomy is almost always fatal in this disease, generally from hæmorrhage.

Varieties.—Occasionally in one of the two principal types of leucocythæmia there may be found in the blood some cells of the kind which usually characterise the other type (*mixed leucocythæmia*).

Acute leucocythæmia is generally of the lymphatic type, though there may be but little enlargement of the glands. It sometimes begins with chill and fever. There may be stomatitis, with swelling of the gums and purpuric hæmorrhages. The duration is ordinarily from a week to two months.

Chloroma (*green cancer*) is a rare disease which appears to occupy a position midway between leucocythæmia and lymphosarcoma. There is a tumour-like change in the bone-marrow, and lymphoid growths of a greenish colour develop in such regions as the orbit, the cranial bones, the spine, and the skin. The cause of the green colour is not known. In some of the cases, but not in all, the blood is altered, as in leucocythæmia. Pallor, weakness, and sometimes exophthalmos are among the symptoms. The disease always ends fatally.

vii. Hodgkin's Disease

(Lymphatic Anæmia. Lymphadenoma. Pseudoleukæmia. Adenia. Progressive Multiple Gland Hyperplasia. Malignant Lymphoma).

Classification of Multiple Lymph Gland Enlargements.—The diseases which are specially characterised by enlargement of numerous lymph glands may be arranged under four types.

The first type includes lymphatic leucocythæmia, and also the soft variety of pseudoleukæmia recognised by German writers. Both these affections are characterised by overrunning of the lymph glands, and in some cases of portions of other organs, by lymphocytes; but in lymphatic leucocythæmia the blood contains a great excess of lymphocytes, whereas in the soft variety of pseudoleukæmia it contains no excess, or only a slight excess, of those cells.

The second type is of tubercular origin, and is characterised by progressive enlargement of one group of glands after another. Fibro-cellular connective tissue, multinucleated cells, and frequently tubercle bacilli, can be recognised in these glands. The course is chronic, with little tendency to caseation.

The third type is Hodgkin's disease, or lymphadenoma, and is apparently identical with the hard variety of pseudoleukæmia recognised by German writers. Muir dissents from the view that it is a late stage of the first type; and he and other recent observers dissent also from the opinion, which has been extensively held, that it is really tubercular, and therefore properly included under the second type. Many inoculations on animals with material from such cases have failed to produce tuberculosis or any other disease. Nevertheless, tuberculosis is occasionally added to Hodgkin's disease as a secondary infection.

The fourth type includes tumours taking origin in lymph glands (lymphosarcoma), the most common being that which arises in connection with the mediastinal and bronchial lymph glands, and spreads along the bronchial lymphatics. This disease is rarely associated with a general enlargement of the lymph glands, but when it is, the case is clinically indistinguishable from one of the second or of the third type. Cases of the fourth type, however, may be, histologically, not lymphosarcoma, but spindle-celled sarcoma or endothelioma.

It is noteworthy that these diseases, whether infective or neoplastic (second, third, and fourth types), having taken origin in lymph glands, continue to show a special tendency to involve lymph glands and channels, including in some instances the serous membranes.

Definition of Hodgkin's Disease.—A disease characterised by widespread enlargement of lymph glands, enlargement of the spleen, and anæmia.[1]

Etiology.—All ages are liable. Males suffer more than females. In a few cases, chronic irritation of some tissue or organ has caused enlargement of the related lymph glands, after which other glands have become involved ; but it is seldom that any cause can be traced. The immediate cause is unknown, but the histological changes suggest that it is a chronic infection.

Morbid Anatomy.[2]—The glandular enlargement usually begins in the cervical region, although in a few instances it seems to begin in the retroperitoneal glands, whence it spreads to the mesenteric and inguinal glands. The glandular disease may be almost universal, but as a rule the upper part of the body suffers most. In the early stage the glands are somewhat soft, but at a late period they may be very dense, and have some fibrous matting around them. Section of the glands may reveal necrotic foci, but not caseation. The Malpighian bodies of the spleen are in some cases similarly involved, and growths with the same kind of structure may be present in such parts as the liver, kidneys, intestinal mucosa, and peribronchial tissue.

The histological change is not an overgrowth of lymphoid tissue, but a result of reaction to some irritant, chiefly on the part of the stroma. The connective-tissue cells and endothelial cells proliferate, as in any other chronic inflammation, and by their numbers tend to conceal the lymphoid tissue. In many cases numerous eosinophile cells are present in the new-formed tissue. As induration supervenes the fibrous elements increase in amount, while the cells become fewer. The density of the new tissue may

[1] The general recognition of this disease was due to Hodgkin's paper published in 1832, but cases had been described before that date.

[2] The following account of the morbid histology, as well as the above classification, is based on Muir's description ('On Leucocythæmia, Lymphadenoma, and Allied Diseases,' *Glasg. Med. Jour.*, 1905, lxiv., pp. 161-174).

interfere with its nutrition, and lead to degeneration and even necrosis. The bone-marrow is unaltered, or may show some increase of the red marrow.

Symptoms.—Enlargement of a group of superficial glands, especially the cervical, is usually the first symptom. Occasionally, however, attention is first attracted by the anæmia, or by pressure-effects referable to enlargement of deep glands. The swellings are painless, and may grow into large tumours which cause serious trouble and even death by compressing the trachea, pharynx, gullet, veins of the neck and limbs, nerves, bile-ducts, etc. The recurrent nerves may be compressed by intrathoracic tumours, and the nerves of the limbs by masses in the axillæ and groins. Enlarged retroperitoneal glands may compress the solar plexus and cause symptoms suggestive of Addison's disease. The spleen is in most cases enlarged, but not to an extreme degree.

Anæmia always supervenes, and is of the secondary type. The hæmoglobin may be reduced by half ; the red corpuscles not quite so much. Poikilocytes and normoblasts are sometimes present. The leucocytes are not as a rule increased, but occasionally there is an increase of polynuclear neutrophiles. There is usually fever, which may be continuous, hectic, or irregular. Shortness of breath, palpitation, dropsy, emaciation, and increasing weakness are also noteworthy. The pulse is accelerated and soft. Epistaxis and diarrhœa may be symptoms. The dyspnœa, due in part to the anæmia, may be due also to pressure on the air-passages ; and similarly the dropsy, due largely to anæmia and cardiac weakness, may be due in part also to pressure on veins.

Diagnosis.—The distinction from tuberculosis may be difficult in the early stages when the swelling is confined to one group of glands. Moreover, even with glandular enlargements in two, three or four different regions in an adolescent, tuberculosis is far more likely than Hodgkin's disease. Tubercular glands commonly tend to caseate, and often, but by no means always, suppurate. The tuberculin test may be employed if the patient is free from fever. It may be desirable to remove a mass for examination.

Lymphatic leucocythæmia is distinguished by the lymphocytosis. Any leucocytosis in Hodgkin's disease is generally due to polynuclear cells.

In *lymphosarcoma*, the disease tends to produce a large mass of glands welded together, and to invade neighbouring structures. It may be advisable to remove a piece of the growth for microscopical examination.

Prognosis.—The course of the disease is very variable, but it almost always ends fatally—usually by asthenia, but sometimes by a pressure-effect. Acute cases may cause death in a few months. In other cases, the disease may remain in its original seat for months or years, and then spread to other parts, sometimes advancing, sometimes stationary, and sometimes even receding; but recovery rarely takes place.

Treatment.—If the growth is localised and anæmia is trifling, the glands might be removed, but the results thus far have not been so good as might be expected. Other local measures are not of much use, though the X rays should be tried. Arsenic is the most important drug, and should be given in as large doses as can be tolerated. Phosphorus is less useful. Cod-liver oil, iron, quinine, and change of air may be recommended. Morphine may be required for pressure-pains.

viii. Splenic Anæmia (Primary Splenomegaly).

Definition.—A condition characterised by enlargement of the spleen and progressive anæmia.

Etiology.—The anæmia is supposed to be the result of the changes in the spleen. The cause of the disease is unknown, but intestinal infection has been suggested.

Morbid Anatomy.—The spleen is much enlarged. Its capsule is thickened. Its fibrous tissue is increased, and the Malpighian bodies and pulp are atrophied. The organ contains immense numbers of large nucleated cells which enclose red blood corpuscles.

Symptoms.—The onset is insidious. The patient becomes weak and anæmic. He has attacks of pain in the region of the spleen, and this organ is found to be enlarged and

tender. The red corpuscles are reduced to about 60 per cent. of the normal, and the hæmoglobin is reduced about twice as much. The colour index may be lower than in any other known anæmia. The leucocytes are not increased, and may indeed be diminished. The lymph glands are not enlarged. Pyrexia is common. Hæmorrhages may occur from the mucous surfaces, and the skin may become pigmented.

Diagnosis.—Examination of the blood differentiates the disease from medullary *leucocythæmia*. In *Hodgkin's disease* the lymph glands are enlarged. Various other forms of splenic enlargement must be excluded by the history of the case and the associated phenomena.

Prognosis.—In some cases, fair health is preserved for years. The course is protracted, and extends over years. Death results from asthenia, hæmorrhage, etc.

Treatment.—Splenectomy has frequently been successful. Otherwise no satisfactory treatment is known.

The rare condition known as *Banti's disease* is regarded by some as a late stage of splenic anæmia. It is characterised by the splenic enlargement and progressive secondary anæmia of primary splenomegaly, with the addition of secondary multilobular cirrhosis of the liver, jaundice and ascites.

Chronic Polycythæmia with Cyanosis and Enlarged Spleen.

This is a rare condition to which attention has recently been drawn, especially by Osler. There is great increase in the red corpuscles, which may number more than twice the normal. The cyanosis cannot be accounted for by emphysema. The spleen is usually enlarged, and headache, giddiness, and constipation are other common features.

Infantile Splenic Pseudoleukæmia.

This is not to be regarded as a distinct disease. It occurs in children, and is recognised as being specially associated with rickets and syphilis. In well-marked cases, the spleen is greatly enlarged, and anæmia is present. The

red corpuscles are diminished, and poikilocytes and normoblasts are observed. The white corpuscles are increased in number.

Under treatment by tonics, such as cod-liver oil and syrup of the iodide of iron, recovery generally takes place.

ix. Status Lymphaticus (Lymphatic Constitution. Lymphatism).

Definition.—A condition met with chiefly in children, characterised by hyperplasia of the thymus, spleen, lymphoid bone-marrow, lymph glands, and other lymphoid tissues, and frequently associated with hypoplasia of the heart and aorta, and with rickets.

Morbid Anatomy.—The thymus is large and soft. The splenic enlargement is not great; the Malpighian bodies may be distinctly hypertrophied. The yellow marrow of the shafts of long bones may be replaced by red lymphoid marrow. The lymphoid tissue of the alimentary tract—in the tonsils, naso-pharynx and small and large intestines—may be considerably overgrown. The thoracic and abdominal lymph glands are affected more than the superficial glands. The heart and aorta are poorly developed.

Symptoms.—Enlargement of the lymphoid tissues, a feeble circulation, signs of rickets, and a fat, flabby condition of body, are the principal evidences of the existence of lymphatism. The importance of the condition depends upon the fact that children who suffer from it are liable to die from what appear to be quite inadequate causes, such as the administration of an anæsthetic for the removal of adenoids, or from no obvious cause at all. It has been supposed that the sudden death in the latter group of cases is due to pressure upon the trachea by the enlarged thymus, but the post-mortem evidence is frequently opposed to this view. Another theory is that the condition is a toxæmia, due to excessive production of the internal secretion of the thymus.

Treatment.—Abundance of fresh air and sunshine. Syrup of the iodide of iron and arsenic may be recommended.

x. Hæmophilia (Hæmorrhagic Diathesis).

Definition.—A constitutional condition, often congenital and inherited, and characterised by immoderate bleeding, either spontaneously or after slight wounds, and sometimes by obstinate swellings of joints.

Etiology.—Males suffer far more than females. The affection is often inherited. In one American family cases have occurred during two centuries, extending now to the seventh generation. The daughters of a 'bleeder' remain healthy themselves, but are almost sure to transmit the disease to their male offspring. Male bleeders seldom or never transmit the disease to their sons.

Morbid Anatomy and Pathology.—There is no constant change. Hæmorrhages in and around the joints have been observed, and occasionally synovitis. The disease has been supposed to depend on abnormal delicacy of the bloodvessels and imperfect coagulability of the blood. Delay in coagulation is sometimes very marked.

Symptoms.—The tendency to bleed is generally discovered within the first three years of life, but seldom in early infancy. An insignificant cut, a tooth extraction, vaccination, or leeching is followed by serious and persistent hæmorrhage. Serious bleeding takes place from the mouth and other mucous membranes, but especially from the nose. Blood may be effused into the skin and deeper tissues, either spontaneously or after injuries. The blood comes from the capillaries. Profound anæmia may ensue, or death may result after a period varying from hours to weeks. The knee and other large joints may be the seat of painful swelling, suggestive of arthritis, and probably due to hæmorrhage into the synovial cavity; this may be accompanied by pyrexia.

Diagnosis.—This depends on the persistent tendency to serious and prolonged hæmorrhage from different parts, with little or no occasion. The diagnosis is the more certain if the patient is a male, and has inherited the tendency.

Prognosis.—Many patients die in childhood. Tooth-extraction often, and leeching, vaccination, and other trivial

operations occasionally have caused death. Death, however, is rarely due to the first hæmorrhage, and with care even old age may be attained. Menstruation and parturition are not specially dangerous.

Treatment.—Preventive measures are important. Bleeders should be specially protected from injury, and should undergo no operation that is not essential. An occasional saline laxative may help to prevent spontaneous hæmorrhage. If an operation is necessary, the patient should for two or three days beforehand take large doses of calcium (*e.g.*, calcium chloride, 10 to 20 grains thrice daily), so as to increase the coagulability of the blood.

If bleeding has set in, local compression should be tried, and solution of adrenalin chloride (1 in 1,000), or solution of calcium chloride (1 per cent.), or solution of ferric chloride, may be applied as a styptic. Tonics should be given in convalescence.

Bleeders and daughters of bleeders should not marry.

xi. Purpura.

Definition.—A condition characterised by the spontaneous occurrence of hæmorrhages into the skin, and sometimes also by hæmorrhages from mucous membranes.

Morbid Anatomy and Pathology.—The cutaneous hæmorrhages are generally in the form of *petechiæ*, which are little brownish-red spots resembling flea-bites. Sometimes they are in patches (*ecchymoses*), or linear (*vibices*). The discoloration is not removable by pressure, and the tint gradually changes, as in the case of a bruise. After death, petechiæ are often found in the serous membranes, and larger hæmorrhages may be found elsewhere. In Henoch's purpura there may be acute nephritis.

Purpura is probably due to a poison circulating in the blood and damaging the walls of the minute vessels. This poison may be elaborated by micro-organisms, or may be produced in some other way, and accordingly it would appear that purpura is sometimes a disease and sometimes only a symptom. Several varieties may be distinguished.

Symptomatic Purpura.—Purpura is very common in the late stages of Bright's disease, tuberculosis, and other debilitating conditions; the petechiæ are most common on the legs. It occurs also in infectious diseases such as pyæmia and ulcerative endocarditis, in grave forms of measles and smallpox, and as a regular feature of typhus. It may follow the use of certain drugs (*e.g.*, potassium iodide), and it is sometimes observed in hysteria and in structural disease of the spinal cord. Such terms as cachectic, infectious, toxic, and neurotic, have been applied to these different varieties of symptomatic purpura.

Purpura Simplex.—One or more crops of petechiæ appear on the legs and sometimes on the trunk, chiefly in young subjects. There may be no symptoms, or there may be slight articular pains,[1] pyrexia and diarrhœa for a few days.

Purpura Hæmorrhagica (*morbus maculosus* of Werlhof). —This disease is like a further and more severe stage of simple purpura. Beginning like the latter with petechiæ, it becomes after a few days associated with serious and sometimes uncontrollable hæmorrhage from mucous surfaces, such as those of the nose, mouth, urinary tract, uterus, etc. Hæmorrhage may occur in the retina or brain. Profound anæmia is the result. There is little or no fever. In favourable cases the bleeding ceases after a week or two, and the anæmia is gradually recovered from, but death frequently results from exhaustion or from intracranial hæmorrhage. In rare cases of great severity (*purpura fulminans*), the hæmorrhages cause death within a day or two.

It is important in diagnosis to exclude *scurvy*, in which the gums and calves are affected; to exclude *leucocythæmia*, by examination of the blood and by the history of the illness; and to exclude malignant types of the *specific fevers*, which are likely to occur in epidemics.

Purpura Rheumatica (*peliosis rheumatica*, *Schönlein's disease*, *arthritic purpura*, *purpuric erythema*).—Various opinions have been held as to the nature of this disease,

[1] Cases of purpura with involvement of joints (*e.g.*, *P. simplex* and *P. rheumatica*) have been classified as *arthritic purpura*.

and while by most writers it is regarded as a species of purpura, it has been looked upon by others as allied to scurvy, to rheumatism, or to erythema multiforme. For the two last views in particular there is much to be said. The disease may be regarded as one of the types of erythema multiforme which may be manifestations of the rheumatic infection.

Purpura rheumatica is characterised by pains and occasionally swelling in the knees, ankles, and other joints, and by a purpuric eruption which chiefly involves the lower limbs. There may be sore throat and slight pyrexia. A bulla is sometimes seen about the malleolus (pemphigoid purpura), and in some instances wheals and œdema appear (*purpura urticans*). The attack usually passes off in a few weeks, but recurrence is common. Cardiac lesions are quite exceptional, and death is rare.

Henoch's Purpura.—At first there are arthritis and a purpuric eruption as in purpura rheumatica, but to these there is soon added abdominal pain, with vomiting and constipation, followed by diarrhœa. Hæmorrhages take place from the mucous membranes. There may be acute nephritis and enlargement of the spleen. Recurrences may take place during a long period, but ultimate recovery is the rule. The disease occurs chiefly in children.

Treatment of Purpura.—This includes rest in bed ; salicylate of sodium for arthritis ; opiates for severe abdominal pain ; ergot, calcium chloride, turpentine or adrenalin for severe hæmorrhage ; and iron, arsenic, and quinine for anæmia and debility.

xii. Scurvy (Scorbutus).

Definition.—A constitutional condition induced by prolonged deprivation of fresh food, and characterised by general debility, anæmia, swelling of the gums and hæmorrhages.

Etiology.—Scurvy was formerly a deadly foe to sailors on long voyages, and is still occasionally seen in seaport hospitals. It may break out on land among people who, owing

to famine, or through being employed in districts remote from towns, are deprived of fresh food for long periods. In parts of Russia it is endemic and sometimes epidemic. It occasionally occurs in Glasgow and neighbouring places in individuals who, through want of means, or from taste, live on an unsuitable diet. In one such case the daily dietary consisted of about nine slices of loaf bread, weighing, say, 18 ounces, with from 3 to 5 pints of tea (containing milk). and very occasionally one egg. The disease has been thought to be due to a defective supply of potassium salts or of organic salts ; or to diminished alkalinity of the blood, resulting from absolute or relative defect of alkaline salts ; or to some poison resulting from decomposition of the food ; or to a specific organism. But these theories are unproved, and the first two, in particular, are very doubtful. Defective hygiene, fatigue, and conditions which cause physical and mental depression favour the outbreak of scurvy.

Morbid Anatomy and Pathology.—The red corpuscles of the blood are diminished, but there is no leucocytosis. Hæmorrhages are found in the skin, mucous membranes, muscles, and internal organs. The spleen is large and soft, and the parenchyma of the heart, liver and kidneys is degenerated.

Symptoms.—The onset is insidious, with progressive loss of bodily and mental vigour. The skin is pale and sallow. The gums are swollen, spongy and painful, and bleed readily. The teeth are loosened, so that chewing is interfered with or prevented. The breath has an offensive smell. Firm, tender swellings develop in the calves, owing to hæmorrhages in the muscles and subcutaneous tissues. Petechiæ, and perhaps larger hæmorrhages, appear in the skin ; and in severe cases there may be hæmorrhages from mucous membranes, or under the periosteum of bones. Emaciation, and the various symptoms of anæmia, such as breathlessness, palpitation, and œdema, may be observed. There is no pyrexia. Nyctalopia (night blindness) is a well-known symptom of scurvy, though not peculiar to this disease ; it is attributed to exhaustion of the retina. The patient sees well in good daylight, but in poor light, whether natural or

artificial, his vision is much less acute than that of healthy persons.

Complications.—These include pneumonia, gangrene of the lung, pericarditis, and pleurisy. The two latter may be hæmorrhagic. Diarrhœa or dysentery may be present, possibly as a coincidence. In a scorbutic subject, wounds, ulcers, and fractures do not heal properly, and, indeed, a partially healed fracture may become disunited.

Diagnosis.—This depends upon the dietetic history, the state of the gums and the state of the calves.

Prognosis.—If appropriate treatment is available, the prognosis is good, unless the case is advanced. Death may result from exhaustion, intracranial hæmorrhage, pneumonia, pulmonary gangrene or inflammation of serous membranes. When scurvy is recovered from under proper treatment, it leaves no sequel, except an increased susceptibility to the disease when the conditions that cause it arise again.

Treatment.—The disease is prevented by supplying sailors with a daily sufficiency of vegetables or lime-juice. Ordinary cases of the disease readily yield to treatment by perfect rest, fresh meat, green vegetables, and 3 or 4 ounces daily of well-diluted lime or lemon juice, sweetened to taste. If the gums are so painful and the teeth so loose that chewing is impossible, the dietary should be arranged to meet this difficulty; the patient should get milk, eggs, soups and gruel, with mashed potatoes and green vegetables, and minced fresh meat. The mouth should be frequently cleansed with dilute Condy's fluid, or, better, with carbolic acid in boric acid solution.

Infantile Scurvy (*Barlow's disease*, formerly called *acute rickets* and *scurvy rickets*).—The disease is seen chiefly, but not solely, in rickety infants, scurvy and rickets being favoured by similar conditions. One of the most important causes of infantile scurvy is a diet consisting of preserved or manufactured foods instead of breast-milk.

Among the early symptoms there is tenderness of the lower limbs, with pain on movement. More or less symmetrical swellings, due to subperiosteal hæmorrhage, develop

at the epiphyseal junctions, and extend for some distance along the shafts of the femur and tibia especially, though they are sometimes seen in connection with other bones. Crepitation may sometimes be felt at the epiphyseal junctions, owing to separation taking place between epiphysis and shaft. If the child has teeth, the gums may be spongy. The legs are at first flexed, but afterwards flaccid and motionless, as if paralysed. The sternum and costal cartilages may be retracted. There is pallor, and sometimes emaciation.

Recovery takes place under proper treatment. Fresh milk should be used instead of condensed milk and artificial foods. Potato which has been thoroughly boiled and mashed, and then mixed with milk till it is of a creamy consistence, makes an excellent food. Soup or beef-tea in which vegetables have been boiled should also be given, and small quantities of lime-juice, diluted and sweetened, may be administered several times a day.

xiii. Hæmoglobinuria.

Hæmoglobinuria is the presence in the urine of the colouring matter of red blood corpuscles, without these corpuscles themselves. In a well-marked case, the urine has a dark reddish-brown colour, which is due to a mixture of methæmoglobin and oxyhæmoglobin. A considerable sediment is thrown down on standing, and this is found microscopically to consist mainly of granular hæmoglobin, together with tube casts and crystals of calcium oxalate. Occasionally a few red corpuscles may be detected, and the escape of these is to be attributed to the renal irritation and congestion occasioned by the hæmoglobin.

Urine of this kind may be suspected from its colour taken along with the naked-eye appearance of the sediment. It is highly albuminous, and gives the usual reactions for albumen. It yields the guaiac reaction for blood, and in the spectroscope it yields the absorption bands of the two varieties of hæmoglobin, viz., oxy- and methæmoglobin. The distinction from hæmaturia lies in the absence, or almost complete absence, of blood corpuscles.

The hæmoglobinuria is the immediate result of hæmoglobinæmia, the red corpuscles being dissolved in the circulating blood so that their hæmoglobin is set free. The kidneys in this condition have a dark chocolate colour, and their tubules contain granular pigment which has been excreted by way of the Malpighian tufts. These changes are the result of the hæmoglobinæmia. The latter is not due to kidney disease.

Hæmoglobinuria may be either (1) symptomatic or secondary ; or (2) to all appearance primary.

1. Symptomatic Hæmoglobinuria arises in various ways. Thus it can be produced experimentally by injecting the blood of an animal into the circulation of one belonging to a different species ; the corpuscles cannot survive in the foreign blood. Hæmoglobinuria results from poisoning by potassium chlorate, carbolic, hydrochloric and other acids, and the inhalation of arseniuretted hydrogen gas. Extensive burns, and certain infections such as malaria, scarlet fever, and yellow fever may produce it. Even the presumably primary form seems sometimes to be a sequel of malaria, and certainly individual attacks may be due to definite causes, such as exposure. *Epidemic hæmoglobinuria* of new-born infants (*Winckel's disease*) is probably an acute infection. It sometimes appears in epidemic form in maternity hospitals, and is very fatal.

2. Paroxysmal Hæmoglobinuria (formerly called *intermittent hæmatinuria*) is an apparently primary affection which occurs in certain individuals in a paroxysmal manner. Malaria appears to be in some instances a cause of this disease, just as it is of Raynaud's disease, which is sometimes associated with paroxysmal hæmoglobinuria. Syphilis is also regarded as a cause. Exposure to cold, or severe effort, or violent emotion, may excite a paroxysm (as in the case of Raynaud's disease[1]) ; and susceptible persons are liable for an indefinite period to suffer from an attack whenever they are chilled. Males suffer far more than females.

The paroxysm is associated with the disintegration of red

[1] Other points of resemblance between these two disorders are given in the author's monograph on 'Raynaud's Disease' (1899), p. 145 *et seq.*

corpuscles in the blood and the liberation of their hæmoglobin, which is thereupon excreted by the kidneys. The urine which is passed shortly after the attack sets in is dark reddish-brown from the presence of hæmoglobin, as already described. It becomes clear again some hours after the attack ceases. The symptoms include a feeling of cold, pallor, anorexia, nausea, abdominal or lumbar pain, a desire to urinate and defæcate, and jaundice. The attack may last for some hours or for a day or two. There may be a transient rise of temperature, and the number of red corpuscles in the blood is naturally reduced for a time. Recurrence is paroxysmal, but not periodic. Microscopic examination of the urinary sediment makes the diagnosis certain.

Prognosis.—This is good, since the disease does not shorten life (unless doubtfully by setting up chronic changes in the kidneys) ; and if the patient avoids the known causes of attacks, he may in time become free from the liability.

Treatment.—When an attack comes on, or is likely to come on, the patient should promptly take a hot drink and get into a warm bed. A few grains of quinine may also be taken at the outset by an adult. In the intervals, warm clothing, avoidance of exposure, and a quiet life are indicated ; and if malaria or syphilis is a possible cause, the appropriate remedies should be employed.

DISEASES OF THE DUCTLESS GLANDS.

i. Myxœdema (Athyrea. Gull's Disease).

Definition.—An affection depending on loss of function of the thyroid gland, and characterised by a myxœdematous state of the subcutaneous tissues with physical and mental lethargy.[1]

Etiology.—The disease is most common between thirty and fifty years of age, and affects women six or seven times as often as men. Excessive child-bearing seems to predispose.

[1] The symptoms were first described by Sir William Gull in 1873 as those of 'a cretinoid state supervening in adult life in women.' Gull expressed the opinion that the condition was allied to the sporadic cretinism already described by Fagge.

It is occasionally inherited, and may occur in several members of a family. It depends upon loss of function of the thyroid gland associated with fibrosis, but it is not certain whether in the fibrosis atrophy of the cells or overgrowth of the fibrous tissue comes first. The cause of the fibrosis is unknown. Quite exceptionally the destruction of the thyroid tissue has been due to a plainly recognisable cause, such as syphilis or actinomycosis. Occasionally the condition has been preceded by goitre or exophthalmic goitre.

Morbid Anatomy.—The thyroid gland is generally reduced in size, though occasionally enlarged by excess of fibrous tissue or cysts. In any case, the secreting tissue is wasted. In the early stages, there is round-celled infiltration of the alveolar walls, and the epithelial cells show signs of proliferation. The condition gradually passes into fibrosis, with thickening of the walls of the arteries and diminution or occlusion of their lumen. The skin is swollen and translucent, and may contain an increased quantity of mucin (whence the name myxœdema proposed by Ord). The subcutaneous fat is usually abundant. Fibrosis takes place around and in the hair follicles and the sudoriparous and sebaceous glands, thus accounting for the loss of hair and dryness of the skin. But it is noteworthy that even after the disease has lasted for many years, proper treatment will restore the skin with its sweat glands and hair follicles to a normal state.

The hypophysis cerebri is occasionally enlarged.

Symptoms.—The onset is usually gradual. The skin of the face becomes swollen, the lips are thickened, and the eyebrows raised. The malar prominences are flushed, but the rest of the face is pale. The tongue is large. The hands and feet are broad. The body as a whole is increased in bulk. The skin is dry, and, though swollen, does not pit on pressure. There is little or no perspiration. The hair becomes scanty. Movements, as in walking, are performed slowly. Speech is slow and the voice thick. The intellectual processes generally are sluggish. Memory is impaired. The temperature is subnormal. The urine is non-albuminous, and ex-

amination of the different viscera yields negative results. There may be a tendency to fall if the eyes are closed. The patient suffers much more in cold than in warm weather. In late stages, serious mental symptoms may supervene, so that the patient is removed to an asylum. Even without treatment, life may be prolonged for many years, and death is generally from an intercurrent affection.

Diagnosis.—The symptoms of the fully developed disease are quite characteristic. The absence of albuminuria, the absence of pitting of the skin, and the malar flush, are important as excluding Bright's disease. If any doubt still exist, the results of thyroid treatment should be observed.

Prognosis.—By the aid of thyroid extract properly employed, it may be expected that the symptoms of the disease will be removed and prevented from recurring.

Treatment.—Five grains of the official thyroideum siccum or 5 minims of the official liquor may be taken once a day to begin with, and more frequently, after a time, if this should prove to be necessary. After the symptoms are removed, a similar dose may be taken once or twice a week to prevent their return. It is important to avoid overdosage, as this may cause serious cardiac and other symptoms. The patient should always be well protected from cold.

CRETINISM is the result of loss of function of the thyroid gland when this loss is of congenital origin or sets in before puberty. Cretinism is *endemic* in Switzerland and elsewhere, and many cretins have goitre. Cretinism is common in the children of goitrous parents. *Sporadic* cretinism is sometimes seen in this country, and may be due to congenital absence of the gland, or to atrophy following an acute fever or connected with goitre. Intemperance and insanity in the parents, and fright sustained by the mother during pregnancy, are believed to be predisposing causes. The condition is seldom recognised till the child is more than six months old. Bodily growth, including ossification of the bones, and mental development are greatly retarded. Teething, speaking, and walking are greatly delayed. The face is bloated, the skin dry and rough, and the hair scanty. The tongue often hangs from the mouth. The abdomen is

large, and there is sometimes umbilical hernia. Where cretinism is endemic, all degrees of the condition can be recognised, some of the sufferers being able to work, while others are speechless and helpless idiots.

Treatment.—Endemic cases ought to be removed to a healthier locality. The treatment for either class includes 1 grain daily of dry thyroid, or 1 minim of the solution, the dose to be increased if necessary. Chemical food should also be given. The diet should be rich in animal matter. Warm baths, friction of the skin, and careful educational methods are indicated.

Operative Myxœdema or *cachexia strumipriva* is the condition resembling myxœdema which sometimes follows complete extirpation, and much less frequently partial extirpation, of the thyroid gland. The fact that it does not always follow the operation is probably to be explained by the existence of accessory thyroids.

ii. Goitre

(Bronchocele. Thyreocele).

Goitre is a general term for enlargement of the thyroid gland. New growths are not included in this account, and exophthalmic goitre will be separately described.

Etiology.—Goitre may be *sporadic* or *endemic*. The endemic form prevails in many countries, and is exceedingly common in parts of Switzerland. The affection is specially frequent among the inhabitants of deep valleys, but it is not confined to such districts. In this country, it has received local names, such as Derbyshire neck and Lanarkshire neck. According to a recent report, from 200 to 300 cases were connected with one Lanarkshire community. In Switzerland, it is often associated with cretinism. In Britain women are much more frequently affected than men, but in India the two sexes are equally susceptible. Goitre is very often first noticed about puberty.

The immediate cause of the disease is not known. It has been attributed to the drinking of snow-water, to iron, copper, lime or magnesium salts in the water, and to infec-

tion by an organism. Water can certainly be the means of communicating the disease, and certain wells on the Continent have actually been frequented with this purpose in view by men who wished to escape military service. As the water is rendered harmless by boiling, the idea of infection seems well grounded, and Grasset has described a hæmatozoon which he has found in the blood. He suggests that enlargement of the thyroid results from this endemic infection, just as splenic enlargement results from malaria, another endemic infection.

Morbid Anatomy.—The goitre may be (1) simple or parenchymatous, consisting merely in hypertrophy of all the tissue elements ; (2) cystic or colloid, due to great increase in size of the vesicles, some of which may coalesce ; (3) fibrous, in which the connective tissues undergo hypertrophy, and cause atrophy of the vesicles ; or (4) pulsating or vascular, in which the vessels are dilated, as in exophthalmic goitre. Many goitres are really due to adenomata developing in the thyroid.

Goitre may involve the thyroid uniformly, or it may be confined to one lobe or to the isthmus.

Symptoms.—A goitre is accessible to inspection and palpation. It moves upwards when the patient swallows. A large cyst may yield fluctuation. A small goitre causes practically no symptoms, and even a large one, though giving rise to a sense of dragging and discomfort, often causes no serious results. Occasionally, however, there are symptoms of pressure on the trachea, gullet, veins, or recurrent nerves, and sudden death may occur.

Prognosis.—The outlook is favourable. Death from goitre is rare. Spontaneous cure may follow removal from the locality where the disease was acquired. The symptoms of exophthalmic goitre occasionally supervene on ordinary goitre. Suppuration may occur in the goitre. Cretinism is common in the children of those who have endemic goitre, and is itself sometimes associated with that disease.

Treatment.—The patient should be removed from the endemic locality, and if that is not possible, the drinking-water ought to be boiled before use. It is supposed that

simple hypertrophy of the thyroid in adolescents is the expression of an increased need of the secretion of the gland by the body, and therefore the thyroid treatment should be tried as in cases of myxœdema. Another remedy is iodine, painted externally and taken internally, in the latter case as potassium iodide or tincture of iodine. As thyroid secretion contains iodine, the old-standing reputation of iodine is perhaps well grounded. Good results are obtained by the daily inunction of biniodide of mercury ointment over the goitre ; in India the patient is then made to expose his neck for some hours to the sun's rays. Injections of iodine into the gland were formerly practised, but are dangerous. Pressure-symptoms may call for surgical intervention—*e.g.*, removal of the isthmus or of some other part of the gland.

iii. Exophthalmic Goitre

(Graves's Disease. Basedow's Disease. Parry's Disease).

Definition.—A disease characterised by cardiovascular disturbance, exophthalmos, goitre and muscular tremor.[1]

Etiology.—About 95 per cent. of the cases are females. The disease may occur at any age, but it begins most frequently between twenty and thirty. It is predisposed to by a neuropathic heredity, debility and anæmia. Quite exceptionally it is inherited, or occurs in several members of a family (*e.g.*, mother, son and daughter). The most frequent exciting cause is strong emotion.

Morbid Anatomy.—The heart is dilated and hypertrophied. The arteries in the region of the diseased gland are dilated. Sometimes the fat in the orbit is increased. No significant change is found in the central nervous system, cardiac ganglia or cervical sympathetic. The thymus is persistent and often enlarged. In the enlarged thyroid the colloid matter disappears, and is replaced by a watery material. The acini become racemose instead of circular in type, and their cells tend to become more columnar than in health.

[1] This disease was first fully described by Graves in 1835.

Pathology.—The exact nature of the disease is still obscure. It was formerly looked upon as a disorder of the sympathetic, but this theory does not explain all the facts. Some would probably still regard it as a pure neurosis or functional disorder of the nervous system, but there is a strong tendency now to consider the symptoms as due to excessive function of the thyroid gland (hyperthyrea), and it is indeed possible that the primary seat of disease is this gland itself. In favour of the latter view, it may be pointed out that the phenomena of Graves's disease present a marked contrast to those of myxœdema, which depends upon defect of the thyroid secretion ; that thyroid extract tends to aggravate the symptoms of Graves's disease ; that thyroid extract given in excess for myxœdema causes some of the symptoms of Graves's disease ; and that histological examination of the thyroid in Graves's disease suggests that the gland is in process of active evolution.

Symptoms.—The onset is usually gradual, and in most cases the cardiac disturbance comes first. The heart beats with abnormal frequency and abnormal force. In time it becomes dilated, so that the area of dulness may be increased, and the first sound becomes short as well as loud. A systolic murmur is usually heard over much or all of the cardiac area, but especially at the base. The dilatation may lead to relative incompetence of the mitral valve. The arteries of the neck and thyroid body are dilated, and pulsate with abnormal force. The systolic murmur heard at the base of the heart is continuous with a similar murmur heard over the thyroid.

Palpitation may be present for months or years before the enlargement of the thyroid gland is noted. This goitre or struma sets in slowly, and is painless. It may be symmetrical or irregular, is seldom of great size, and is frequently quite trifling.

The exophthalmos often sets in along with the goitre. It may be slight, or so great that the eyelids do not meet even in sleep. The pupils and ophthalmoscopic appearances are normal. When the eyeball is rotated downwards, the upper lid often follows too slowly, so that a band of sclerotic is

exposed between the upper corneal margin and the upper lid (Graefe's symptom); or there may be retraction of the upper lid with absence of blinking (Stellwag's symptom). The power of convergence is sometimes deficient, through weakness of the internal recti (Möbius's symptom); and other palsies of the external ocular muscles are occasionally observed.

Muscular tremor is generally present, especially in the hands. It varies a good deal in kind and degree, and is best seen when the patient holds her arms stretched out in front of her. The general health is apt to suffer. Emaciation, flushing, sweating, diarrhœa, menstrual disorders, great nervousness increased by attention, insomnia, mental irritability, dyspnœa, vomiting, and cutaneous pigmentation may be present. Glycosuria and albuminuria are not very rare. Œdema of the lower limbs may result from cardiac weakness, but apart from this, œdema may occur in irregular localised patches in various parts of the body without obvious cause. The electrical resistance of the body is diminished, owing to the increased activity of the sweat glands and the dilatation of the bloodvessels. Paraparesis, or sudden giving way of the legs, is sometimes complained of. Hallucinations are occasionally present, and acute mania may develop and cause death.

Acromegaly has been met with in association with exophthalmic goitre.

Diagnosis.—The disorder of the heart and arteries, the exophthalmos, the goitre, Graefe's symptom and the muscular tremors are the leading symptoms. It is to be noted, however, that Graefe's symptom is not confined to this disease. The principal difficulty, perhaps, is in the case of young women who have small goitres but no other signs of the disease, or at most but a slight acceleration of the heart's action. In Graves's disease, the cardiac condition is very important. But it is also important to bear in mind that a large endemic goitre may induce some of the phenomena of Graves's disease by exerting pressure on the cervical sympathetic and possibly on the vagus.

Prognosis.—The course of the disease varies greatly. It usually extends over several years. A certain proportion of

cases (estimated at a fourth) recover. The more severe cases do not recover completely, but the symptoms may after a time subside and cause almost no trouble. Repeated attacks may occur, or one attack may be characterised by a series of exacerbations and remissions. Some cases run an acute or subacute course with or without delirium. In fatal cases, death is commonly due to the cardiac condition and to general weakness, which may be increased by severe diarrhœa. Myxœdema occasionally follows exophthalmic goitre.

Treatment. — This should be carried out as much as possible in the open air. The patient should get abundance of good food. In severe cases, absolute rest of body and mind is important. Drugs are unreliable ; what appears to have done good in one case may be useless in a long series of other cases. One of the most frequently helpful is belladonna given in doses as large as the patient can endure. Another useful drug is arsenic given in small doses for a long time. When the pulse is very rapid tincture of convallaria, of strophanthus, or of digitalis may be added to the arsenic. Bromides may lessen the mental irritability. Opium should be given for diarrhœa, and morphine hypodermically for obstinate vomiting. Sulphonal may be given for some nights as a hypnotic. Suprarenal and thymus extracts are occasionally of service. Galvanisation of the cervical sympathetic, and prolonged application of an ice-bag over the heart or manubrium sterni, may greatly reduce the frequency of the pulse for a time. Very good results are sometimes obtained from the faradic current as recommended by Horsley. Two large electrodes are used, covered with washleather or flannel, moistened with warm salt solution, and applied, one over the goitre, and the other at the back of the neck. The current should not cause pain, and should be applied for from one to four hours daily, at several sittings. The X rays have proved beneficial in a few cases. Operations on the thyroid gland in this disease (*e.g.*, removal of the isthmus, which will arrest the disease if the patient survives) are dangerous, and ought not to be attempted except in severe cases ; under such circumstances only a local anæsthetic should be used. Resection of the sympathetic has been advocated.

On the theory that the function of the thyroid secretion is to neutralise certain harmful substances in the body, it was thought that when thyroid secretion was present in excess, as in Graves's disease, it might be neutralised by introducing into the body blood or serum containing these ordinarily harmful substances. And thus cases of Graves's disease have been treated with blood or serum from thyroidectomised dogs, sheep or goats, or even with blood from a case of myxœdema. *Antithyroidin* is serum from thyroidectomised sheep. The milk of thyroidectomised animals has also been employed, and the dried milk of thyroidless goats is obtainable under the name of *rodagen*. It cannot be said, however, that the results obtained from serum treatment are better than those hitherto obtained by medicinal, electrical, and hygienic measures.

iv. Diseases of the Spleen.

Examination of the Spleen.—Under ordinary conditions of health, the spleen can be examined only by percussion, since it lies wholly under cover of the ribs. It lies beneath the ninth, tenth, and eleventh ribs of the left side, and is separated from them by the diaphragm, and at its upper part by the lung also. It lies obliquely, with its long axis running along the tenth rib. Horizontally the splenic dulness extends from about the posterior axillary to the midaxillary line.

In exceptional cases, it may be justifiable to examine blood taken from the spleen in life by puncturing the organ.

When the spleen enlarges, it does so in a direction downwards and forwards, and it is considerably swollen when it protrudes from under the ribs. Still greater enlargement may cause the organ to extend almost to the pubis and beyond the middle line, in addition to occupying the left flank and iliac region. The situation of the mass, extending upwards as it does under cover of the left lower ribs, its direction of enlargement, and the notched anterior edge are points of distinction from other tumours.

Enlargement of the Spleen is frequently observed in an acute form in febrile diseases. Chronic enlargement is

met with in malarial cachexia, splenic anæmia, leucocythæmia, Hodgkin's disease, cirrhosis of the liver, rickets, and inherited syphilis.

Infarction may result from simple or septic embolism, and probably also from thrombosis. In cases where embolism might be expected, its occurrence may be indicated by pain and tenderness in the region of the spleen, enlargement of the organ, perhaps peritoneal friction over it, and sudden elevations of temperature. There may be evidences of embolism in other organs (*e.g.*, albuminuria or hemiplegia).

Rupture of the Spleen may occur spontaneously, though very rarely, in the acute congestion of malarial or enteric fever. Rupture of the swollen organ may also result from a blow or from an exploratory puncture with a needle. It is followed by bleeding into the abdomen, and calls for immediate operation.

Abscess of the Spleen is occasionally met with. It may be due to the *Bacillus typhosus*, though no intestinal lesions are present.[1]

Movable Spleen or wandering spleen is occasionally met with, chiefly in women. It may be an isolated condition, or may be part of an enteroptosis.

The organ is usually enlarged, and in some cases the increased weight has doubtless been the cause of the stretching of the ligaments, but in other instances the condition appears to be congenital.

There may be no symptoms, or the patient may suffer from a sense of uneasiness or weight. Occasionally, as Osler points out, torsion of the pedicle may lead to great swelling and necrosis, with high fever.

The organ may sometimes be kept in position by a pad and belt, or it may be fixed in place by operation, but occasionally splenectomy may be desirable.

Marked dislocation may occur in the case of a leucocythæmic spleen.

[1] Monro and Campbell, *Trans. Glasgow Path. and Clin. Soc.*, May, 1904; Eve and Thresh, *Med. Press and Circ.*, May 11, 1904.

v. Addison's Disease

(Bronzed Skin Disease).

Definition.—A disease characterised by great muscular weakness, feebleness of the circulation, gastric disturbance, and pigmentation of the skin, and usually associated with tuberculosis of the suprarenal bodies.[1]

Etiology.—Addison's disease is most common between twenty and forty years of age, and is seen chiefly in hospital patients. Males suffer more than females. The disease has sometimes appeared to result from a blow on the back or abdomen. The lesion of the adrenals is occasionally due to extension from tuberculosis of the vertebræ.

Morbid Anatomy.—In 80 per cent. of fatal cases, the suprarenal capsules (almost always both capsules) are tubercular. Caseation, followed by softening or calcification, begins in the medulla and spreads to the cortex, and the inflammatory changes thus induced may involve the neighbouring plexuses and ganglia of the sympathetic. (Tuberculosis, however, frequently attacks these glands without causing any symptoms of Addison's disease.) In exceptional cases there is simple atrophy, or inflammation with atrophy, or secondary malignant disease (primary malignant disease does not cause typical symptoms of Addison's disease) ; or the adrenals may be healthy while the neighbouring semilunar ganglia and other structures are diseased.

The adjacent sympathetic nerves and plexuses are often damaged, but are sometimes healthy. The mesenteric glands may be enlarged. The heart is wasted. The thymus is sometimes persistent. The lungs and other parts may be tubercular, but not uncommonly the suprarenal glands alone are involved in this way.

Pathology.—Two different theories of the disease naturally result from the differing anatomical conditions ; one being that the symptoms result from loss of function of the suprarenal bodies, just as myxœdema results from loss of function of the thyroid ; while the other view is that Addison's disease

[1] See historical note on Pernicious Anæmia, p. 312.

depends on irritation of the sympathetic fibres either in the gland substance or in the neighbouring plexuses. Neither theory is quite satisfactory—the nervous theory even less than the other—and accordingly it has been supposed that sometimes the one and sometimes the other explanation is correct, or, again, that the two causes may co-operate in producing the symptoms. Adrenal inadequacy might mean either loss of the internal secretion of the gland, or accumulation in the system of poisons which the adrenals ought to destroy. Extract of suprarenal capsules has an action almost the same as that of nicotine. It causes powerful contraction of the bloodvessels and great increase in the blood-pressure. Absence of this principle from the blood through disease of the capsules would thus account for the remarkably low tension of the pulse in Addison's disease.

Symptoms.—The onset is usually insidious, with weakness which increases to intense languor and debility. There may be some pain in the epigastric region. Though the nutrition of the body generally and of the muscles in particular may appear good, the muscular feebleness may be extreme. The heart also is feeble, and the pulse extremely compressible. The slightest effort may induce syncope, and this may prove fatal. Anæmia is not a conspicuous feature in this disease.

The pigmentation or bronzing varies in different cases from mere sallowness to dark-brown or almost black. It is specially marked on exposed parts such as the face and backs of the hands ; on parts naturally dark, as the axillæ, areolæ of the nipples, and genitals ; and on parts irritated or compressed by clothing, such as the waist. It involves also the mucous membranes of the mouth and vagina. The colour is due to an increased amount of melanin, the pigment which is normally present in the Malpighian layer of the epidermis.

Nausea, vomiting, and flatulence may be troublesome and obstinate symptoms, and diarrhœa is not uncommon. The temperature is usually normal or subnormal. Death may be due to syncope, asthenia, diarrhœa, or tubercular disease elsewhere.

Diagnosis.—In typical cases, this is easy. Pigmentation of the skin is produced by numerous causes, but the associated phenomena and the history will almost always throw light upon the condition. In particular there may be mentioned the pigmentation of pregnancy and uterine disease (chloasma), that of hepatic cirrhosis with diabetes, deep jaundice, pediculosis, tinea versicolor, and argyria. The weakness of the heart and voluntary muscles, the gastro-intestinal disturbance, and the pigmentation are the most important phenomena, but it should be remembered that the pigmentation may be so slight, even at the time of death, as not of itself to suggest Addison's disease. In such cases, the marked asthenia, the almost imperceptible pulse, and the vomiting, especially if no definite signs of visceral disease are present, should suggest the diagnosis.

Prognosis.—This is almost hopeless, but temporary improvement may occur. The average duration is a few years. In rare cases, recovery takes place.

Treatment.—In a few cases, improvement has followed the administration of suprarenal gland from the sheep (15 grains twice daily, to be increased). Adrenalin may be used instead of the gland itself. If this does no good when given by the mouth, a third of the quantity may be given hypodermically. Fresh air, abundance of light food, and sufficient clothing are important. Tonics, such as arsenic and strychnine, and if possible cod-liver oil, should be given. Symptoms must be treated as they arise—*e.g.*, vomiting by ice and hydrocyanic acid internally, and by a mustard-plaster over the epigastrium ; diarrhœa by bismuth, or by lead and opium. Where distinct asthenia is present, rest in bed should be enjoined, so as to diminish the risk of fatal syncope.

SECTION V

DISEASES OF THE RESPIRATORY SYSTEM

ANATOMY

The apex of the *lung* usually projects for some little distance above the level of the clavicle in front. At the back it reaches up to the level of the seventh cervical spinous process. The anterior border passes downwards and inwards from the apex, behind the sterno-clavicular articulation, and reaches, or nearly reaches, the middle line at the junction of the manubrium with the body of the sternum. The right lung sometimes reaches the left margin of the sternum. From this level, the anterior margin of the right lung continues downwards to the level of the sixth chondrosternal articulation, whereas the anterior margin of the left lung passes from the fourth left chondrosternal junction downwards and outwards to the apex of the heart.

Near the middle line behind, the lungs touch the sides of the bodies of the vertebræ.

The lower border of each lung nearly corresponds to a line, slightly convex downwards, passing round the chest from the sixth chondrosternal joint to the tenth dorsal spine. In the nipple line this will correspond to the sixth rib ; in the anterior axillary line to the seventh ; in the posterior axillary line to the eighth ; and in the scapular line to the tenth rib.

It is to be noted that the *pleural sacs* extend a good deal further down than the lungs.

The boundary between the upper and lower *lobes* of each

lung corresponds to a line drawn from the second dorsal spine to the sixth rib in the nipple line. In the case of the right lung, a line drawn from the middle of the line just described to the sternum at the fourth costal cartilage indicates the upper border of the middle lobe.

The *larynx* ends and the trachea begins on a level with the space between the sixth and seventh cervical spines. The bifurcation of the *trachea* takes place opposite the fourth dorsal spine.

For convenience of description various lines and regions are recognised. The *lines* are : (1) midsternal, (2) lateral sternal, (3) parasternal (midway between lateral sternal and mammary), (4) mammary or nipple, (5) anterior axillary, (6) midaxillary, (7) posterior axillary, (8) scapular (corresponding to the lower angle of the scapula when the arm is hanging by the side), and (9) vertebral.

The *regions* are best indicated by reference to the bones of the chest-wall, but some are defined in a more arbitrary manner. They are as follows :

Anteriorly : (1) suprasternal, (2) sternal, and (3) xiphisternal (infrasternal depression, *scrobiculus cordis*, pit of the stomach) ; (4) supraclavicular, (5) clavicular, (6) infraclavicular, (7) mammary (the last two meeting at the level of the third costal cartilage), and, (8) inframammary (meeting the mammary at the level of the sixth costal cartilage).

Laterally : (9) axillary, and (10) infra-axillary (meeting the axillary at the sixth rib).

Posteriorly : (11) suprascapular, scapular—including (12) supraspinous and (13) infraspinous—and (14) infrascapular ; (15) interscapular (between the scapula and the vertebral line).

EXAMINATION OF THE CHEST.

Physical examination of the chest, or the investigation of those phenomena connected with the thoracic organs which can be studied without the help of the person examined, is carried out by four principal methods, viz., inspection, palpa-

tion, percussion, and auscultation. To these there may be added three subsidiary or combined methods, viz., radiography, mensuration and succussion.

Inspection.

By inspection we study first the form, and secondly the movements of the chest.

The *form* of the chest is accurately studied and recorded by means of the *cyrtometer*, the most convenient form of which consists of two pieces of soft gas-pipe which have been hammered flat and united by a short bit of rubber tubing. The joint is placed over the spine, and the two pieces of metal are moulded to the wall of the chest, a mark being made on each where they cross in front. The instrument is then opened out, and carefully placed on a large sheet of paper on a table. The two halves are then approximated as they were on the chest, marks are made on the paper to indicate the middle line of the front and back, and the circumference is then traced out.

The circumference of the chest is almost circular in the infant, but within a couple of years it has become an ellipse with its long axis transverse, and so it remains, under normal conditions, for the rest of life.

Abnormalities of form may be (1) bilateral, (2) unilateral, or (3) local.

(1) Bilateral abnormalities of form may be indicative of (*a*) a tendency to disease (alar chest and flat chest); (*b*) past disease (pigeon breast, Harrison's sulcus and rickety chest); or (*c*) presently existing disease (emphysematous chest and generally retracted chest).

The *alar* (*pterygoid* or *winged*) chest is so called because the scapulæ project like wings at the back. It is abnormally small, especially as regards its antero-posterior diameter, owing to drooping of the ribs. The drooping of the ribs allows the shoulders to fall, and this in turn accounts for the projection of the shoulder-blades. The smallness of the chest is a congenital condition, and predisposes to phthisis. It is often associated with a long neck and prominent larynx.

The *flat* chest is characterised by great relative deficiency in the antero-posterior measurement, which is due to straightness of the costal cartilages. The sternum is sometimes drawn back to a plane behind the costal cartilages. This deformity, like the alar chest, is congenital, and predisposes to consumption. These two varieties of chest have accordingly been termed *phthinoid*, and individuals who possess them are *phthinodes*.

In the *pigeon breast*, the cyrtometer tracing is triangular, owing to the sternum being thrown forwards, and the ribs straightened out in front of their angles. The condition is due to embarrassment of inspiration when the ribs are soft and yielding. Whooping-cough, measles, bronchitis, and chronic enlargement of the tonsils cause difficulty in inspiration ; and the ribs are soft in childhood, especially if rickets is present. The sternum is drawn forwards by the forced inspirations that are necessary.

Harrison's sulcus, or *transverse constriction* of the chest, extends across each half of the chest in front, at about the level of the xiphoid cartilage. The cause is inspiratory embarrassment in childhood. The muscles of forced inspiration act more powerfully on the upper than on the lower part of the chest, and while the former is being expanded, the latter tends to sink in under atmospheric pressure. But the vault of the diaphragm, supported by the abdominal viscera, prevents the lowest ribs from being pressed inwards in this way, and thus the transverse constriction develops just above the level of the diaphragm.

The *rickety* chest is due to rickets alone. It is characterised by the presence of a vertical groove on each side, near the anterior ends of the bony ribs. The cyrtometer tracing is therefore *fiddle-shaped*. As the circumference of the chest in infancy is circular, respiration is almost purely abdominal, and when the diaphragm is depressed in inspiration, the soft rickety ribs tend to be drawn inwards at their anterior ends.

The *emphysematous*, or *barrel-shaped* chest, is circular in horizontal section, the antero-posterior and transverse axes having become almost equal. This means that the capacity

of the chest has been increased to its utmost, viz., to much greater than normal, by frequent forced inspirations. The sternum is thrown forward and arched with its convexity forwards, and the thoracic spine is arched with its convexity backwards. It should be remembered that caries of the thoracic spine may cause the antero-posterior to equal or even exceed the transverse diameter of the chest.

Bilateral diminution of the chest occurs in phthisis. The thorax then resembles the flat type of congenital deformity.

(2) Unilateral abnormalities of form may be due to either enlargement or diminution in the size of one side. *Unilateral enlargement* may be due either to increase in the bulk of the lung, *e.g.*, by compensatory hypertrophy; or to liquid or gaseous effusion in the pleural cavity. *Unilateral diminution* in the volume of the chest is met with in fibroid phthisis, cirrhosis of the lung and adherent pleura, and in collapse of the lung from obstruction of the main bronchus. In well-marked cases the shoulder of the affected side is drawn down, and the spine is curved with its convexity towards the healthy side. It is therefore necessary, in cases of deformity of the lateral wall of the chest with lateral curvature of the spine, to try and ascertain which is secondary to the other.

(3) Local abnormalities of form consist of localised bulging or shrinking. *Bulging* may be observed in connection with enlargement of the heart, aneurysm, new growths, empyema, etc. Local *shrinking* is often seen over the apex of the lung in phthisis. At the lower sternum it may be due to inspiratory obstruction, or to the pressure of the cobbler's last.

The *movements* of the chest in respiration are repeated about sixteen times a minute in the healthy adult male. The normal ratio between pulse and respiration is about four to one. Respiration is effected on the one hand by the upward and forward movement of the chest-wall, and on the other hand by the depression of its floor. The former movement, constituting the costal type of breathing, is specially marked in females; the latter, constituting the diaphragmatic or abdominal type, is specially marked in adult males and young children. In the adult male, the

girth of the chest at the level of the nipples should be 34 inches at the end of expiration. A deep inspiration should increase it by 2 inches or more.

In disease, the movement of the diaphragm may be diminished or lost through ascites, abdominal tumours, peritonitis, great pericardial effusion, or paralysis of the diaphragm. On the other hand, the action of the intercostal muscles may be impaired or lost through disease of their nerves or of the spinal cord.

In *dyspnœa*, again, the respiratory movements are abnormally forcible, and the accessory muscles of respiration are called into action. Thus inspiratory dyspnœa may be due to a morbid blood state, as in diabetic coma, and is then characterised by an abnormal depth of the inspirations. Or it may be due to mechanical obstruction, as in laryngeal diphtheria, in which case, especially in children, the lower portion of the chest-wall is drawn in under atmospheric pressure whilst the upper part is overexpanded by the powerful muscles of forced inspiration. A noise called *stridor* is often produced at the seat of obstruction by the air which gets past it, and stridulous breathing is therefore an important symptom. In expiratory dyspnœa, expiration is prolonged and laborious, owing to difficulty in expelling the air. This is largely due to impaired elasticity of the lung substance, as in emphysema, but may be contributed to by actual obstruction as in bronchitis and asthma. In some cases of laryngeal disease and of cardiac disease, but especially in bronchial asthma, dyspnœa occurs chiefly in fits or paroxysms.

In severe embarrassment of respiration, the patient often requires to sit up (*orthopnœa*). He may be partly propped up by pillows, or may sit quite erect, or may bend forwards. Sometimes he is relieved by sitting with his legs hanging down, or he may get comfort only by standing and perhaps leaning on the back of a chair.

The peculiar symptom known as *Cheyne-Stokes respiration* is alluded to under the symptomatology of heart disease (p. 259).

Palpation.

The principal signs recognised by this method in connection with the lungs are vocal fremitus, friction fremitus, and rhonchal fremitus. Palpation may also be used to corroborate inspection in studying the movements of the chest.

Vocal fremitus (vocal thrill) is the vibration felt by a hand placed on the chest of a person who is speaking. The vibrations are produced at the larynx and are conveyed to the hand of the examiner by the air in the bronchial tubes, the pulmonary tissues and the chest-wall. In health, its distinctness depends on the depth and loudness of the voice, so that it may be very faint in women and children. In disease it is diminished or abolished by agencies which separate the lung from the chest-wall, such as liquid or gaseous effusion in the pleural cavity. It is generally lost in tumour of the lung, but is preserved and often increased over pneumonic and tubercular consolidations.

Friction fremitus (due to pleural friction) and *rhonchal fremitus* (accompanying a rhonchus or râle) can sometimes be detected by palpation, but the corresponding auscultatory phenomena are of much greater value. Moreover, auscultation may be required to distinguish between rhonchal and friction fremitus.

Percussion.

The art of striking the body so as to produce a sound for diagnostic purposes is probably, as Gee remarks, as old as Hippocrates (say 400 B.C.). It was first employed in abdominal diseases to distinguish ascites from tympanites, and its use was extended to chest affections by Auenbrugger in 1761. Its great value, however, was not generally recognised until Corvisart published a translation of Auenbrugger's essay in 1808.

Percussion is performed by means of a hammer or striker called the *plessor*, and usually also by the aid of a *pleximeter*. In practice, the middle finger of the right hand is the plessor generally employed. The pleximeter may be an oval or oblong piece of ivory or vulcanite which is firmly applied

to the part of the chest examined, but the left middle finger is generally accepted as the best pleximeter for ordinary clinical work. When percussion is made by the medium of the finger or any other pleximeter, it is described as *mediate*. When no pleximeter is used, it is *immediate*. Thus the clavicles are usually struck immediately.

The principle underlying percussion is that a smart tap over an air-filled organ gives rise to a sound in addition to that which is due to the impact ; whereas in the case of a solid organ, any sound produced is due simply to the impact. Percussion in the latter case is dull ; in the former, clear.

Moreover, clear notes are of two kinds : the one drum-like or tympanitic, and the other non-tympanitic. Tympanicity does not depend on the volume of air present, since it may be detected over a cavity an inch or two in diameter, whereas it is not detected over the normal lung owing to the minute subdivision of the contained air.

Percussion notes then are dull or clear, and clear notes are tympanitic or non-tympanitic. Clear notes, whether tympanitic or non-tympanitic, may be of high or low pitch.

By means of percussion we are enabled (1) to ascertain whether a certain portion of the chest yields the normal clear sound, or an abnormal clear sound, or a dull sound ; (2) to delimit an air-filled from a non-air-filled organ (*e.g.*, the lung from the heart or liver) ; and (3) to ascertain the resistance offered by the part (the liver, the heart, a consolidated lung, and a solid or liquid pleural effusion being less compressible and more resistant than a normal or emphysematous lung or a pneumothorax).

Changes in Pulmonary Percussion Note.—*Hyperresonance*, or an abnormal degree of clearness, is observed in some cases of emphysema. Impairment of the clear percussion, in all degrees down to absolute *dulness*, results from consolidation of the lung or from the presence of liquid effusion in the pleural cavity. The normal clear note of the lung is replaced by a *tympanitic* note in pneumothorax and over a large pulmonary cavity. Sometimes a diminution in the volume of the percussion sound is accompanied by a change of quality, so that the note is recognised as *dull-*

tympanitic. Under certain circumstances a variety of percussion note is obtained which is called *subtympanitic*, *tracheal* or *tubular*. It is supposed to resemble the note obtained over the trachea when the mouth is open, and is often met with over the affected portion of lung in certain stages of pneumonia. Another instance of this subtympanitic percussion is the *Skodaic resonance*[1] obtained over the lung above the level of dulness in cases of liquid pleural effusion.

A percussion sound which scarcely lends itself to the above classification is the *cracked-pot sound* (metallic chink), which is obtained by percussion over a pulmonary cavity communicating with a bronchus, if the patient's mouth is open. It may be obtained, however, over the chests of healthy crying children. It may be imitated by clasping the hands loosely, and striking the back of one of them on the knee.

Auscultation.

This method of physical diagnosis was discovered in 1816 by Laennec, who published his great work on mediate auscultation three years later. Immediate auscultation is sometimes useful, *e.g.*, in young children who are frightened by the sight of any instrument ; but as a rule auscultation is mediate, and carried out by the aid of a stethoscope. In the case of the respiratory organs, three kinds of phenomena are studied by this method, viz., (1) the respiratory murmur, which may be changed either quantitatively or qualitatively ; (2) the vocal resonance, which may likewise undergo a quantitative or a qualitative change ; and (3) adventitious sounds or accompaniments, not heard under normal conditions, and including râles and friction sounds.

(1) Two kinds of Breath-Sound can be heard in a healthy individual : vesicular breathing, which is audible over most of the spongy lung substance ; and bronchial breathing, which is heard over the large bronchi and the trachea. The

[1] So named after Joseph Skoda (1805-1881), who was Professor in the University of Vienna from 1846 to 1871. His great ' Treatise on Percussion and Auscultation ' was published in 1839.

vesicular breath-sound is due to the friction of air passing between the finest bronchial tubes and the air-sacs, and also to conduction from the glottis. Under ordinary circumstances, the sound which accompanies inspiration is soft and breezy, has the same duration as the inspiratory act itself, and fades without perceptible interval into the expiratory murmur. The latter is faint in comparison with the inspiratory murmur, and is only perceptible during one-third or even less of the expiratory act. In *bronchial breathing*, the expiratory and inspiratory portions are almost alike in length and in pitch ; they are distinctly separated from one another ; and they have a hollow, blowing quality. The sounds are produced at the glottis, and conducted along the air-passages.

Changes in the Breath-Sounds.—*Enfeeblement of the vesicular murmur* means weak production or weak conduction. The person may be breathing very calmly, or the bronchial tubes may be obstructed ; or there may be a thick layer of fat and muscle outside the chest-wall, or a collection of fluid in the pleura interfering with the conduction to the ear. Enfeeblement occasionally amounts to complete suppression.

Exaggeration of the breath-sounds is known also as *puerile breathing* because it is normal in children, who breathe quickly and have thin chest-walls. In adults who have disease in a limited portion of lung, puerile breathing of a compensatory nature may be observed over the healthy parts.

In *jerky*, *cogwheel*, *interrupted*, or *wavy breathing*, the inspiratory and sometimes the expiratory murmur is broken up into two or more portions. The phenomenon has no clinical importance.

Bronchial breathing replaces vesicular breathing in cases of consolidation and of cavity. On the one hand, destruction or filling up of the vesicles prevents the movement of air into them, and on the other hand, the less spongy condition of the lung tissue favours the conduction of that element in the breath-sound which is produced at the glottis.

Cavernous breathing is bronchial breathing of low pitch and intensely hollow quality. It may be observed over a large pulmonary cavity.

Tubular breathing is bronchial breathing of high pitch and a peculiar whiffing quality. It is chiefly noted over hepatised lung, and points to a lesion at the surface.

Amphoric Phenomena are four physical signs which may be met with in connection with a large cavity containing air, usually a pneumothorax, but occasionally a very large intra-pulmonary cavity. The first two are developed spontaneously ; the last two are elicited by the observer.

Amphoric hum (*amphoric echo*) is metallic resonance similar to that which is perceived on blowing, coughing, etc., into a large empty bottle. It may accompany the breath-sounds, râles, the voice, cough, and the cardiac sounds.

Metallic tinkle is a sound like that produced when a vessel of glass or metal is gently struck with a pin. It may accompany the breath-sound, voice, and cough.

Metallic ring (*bell-sound*, *amphoric note*, *empty bottle note*, *coin sound*, *bruit d'airain*) is the metallic ring of a percussion note as observed by auscultation. Whilst the physician auscultates the chest, an assistant percusses over another part of the cavity, using one coin as a pleximeter and another as a plessor.

Succussion sound (well known to Hippocrates, and therefore sometimes called *Hippocratic succussion sound*) is the splashing sound induced by shaking a mixture of air and liquid such as is contained in the chest in pyopneumothorax.

The first three amphoric phenomena are indicative of a large air-filled cavity ; the fourth requires the presence of liquid in addition.

(2) Vocal Resonance is the voice heard by auscultation. The fundamental tone of the voice is produced at the glottis ; the articulated overtones are generated in the mouth. Just as there are two kinds of breath-sound, so there are two kinds of vocal resonance. *Bronchophony* is the clear sound of the voice as heard on auscultation over the trachea and larger bronchi. The *normal vocal resonance* heard over spongy lung tissue is muffled, and is less loud than that heard over the bronchi, owing to the distance from the

larynx, and still more owing to the spongy character of the conducting lung tissue. Loudness, however, is not the important point, and indeed the fundamental glottic sound may drown the articulated overtones. In such a case the patient should be asked to whisper, and *whispered bronchophony* may then become quite evident. Bronchophony heard over the lung points to consolidation or cavity, and is explained in the same way as bronchial breathing.

Pectoriloquy (breast-speech) is an exquisite degree of bronchophony. The whispered form is particularly striking in pneumonic consolidation. Like tubular breathing, it indicates a surface lesion.

Ægophony is bronchophony with a tremulous, bleating and nasal quality. It is heard principally at the back of the chest in cases where a thin layer of pleural effusion separates the lung from the thoracic wall, and has been attributed to the effusion intercepting the fundamental laryngeal tone more than the overtones or harmonics.

Diminution of vocal resonance is caused by obstruction of a main bronchus, by agencies which separate the lung from the chest-wall (pleural effusion, thickening of the pleura, etc.), and by emphysema. In abundant pleural effusion, and in pneumothorax, the vocal resonance may be abolished.

The study of vocal resonance is chiefly of corroborative value, but it may be important if breathing is very faint or very noisy. Crying in children may be studied like the articulate voice in adults.

(3) Adventitious Sounds (*accompaniments*) are additions to, and not modifications of, the sounds normally perceived by auscultation.

Râles or *rhonchi* are variously classified. According to one method they are divided into dry and moist ; dry râles being subdivided into sonorous and sibilant, and moist râles into mucous and crepitant. Some writers confine the term rhonchi to dry râles, and distinguish moist râles as crepitations. A convenient division, which, moreover, follows the lines of Laennec, is into *dry*, *mucous*, and *crepitant*.

Dry râles are called *sonorous* if of low pitch, and *sibilant* if of high pitch. They are the snoring and cooing sounds

popularly known as 'wheezing,' and are due to local narrowing of the bronchial tubes by mucous secretion. They may be accompanied by rhonchal fremitus.

Mucous râles give the impression of bubbles bursting, and are due to air passing through liquid in the bronchial tubes or in the pulmonary alveoli. The liquid may be mucus or pus, as in bronchitis and phthisis ; blood, as in hæmoptysis ; or serum, as in pulmonary œdema. When the râle is so small as almost to resemble the crepitant râle, it is called *subcrepitant*. Consolidation of the lung tissue makes it particularly definite or articulate, as is observed in the subcrepitant râle of the resolution stage of pneumonia which is known as the *crepitus redux*. *Moist crackles*, or *clicking râles*, or *consonating râles* are sharply defined or even metallic, and are heard over softening caseous matter. Moist râles which lack this clear, ringing quality are sometimes distinguished as *bubbling* or *non-consonating*. The coarse and hollow mucous râles produced in a cavity of moderate size are described as *cavernous* or *gurgling*. Crackling râles are apt to obscure the breath-sounds.

Crepitant râles are not so well understood as the others. They are extremely fine crackling sounds heard chiefly in the later part of inspiration. They have been compared to the sound produced by rubbing the hair between the finger and thumb close to the ear, and to the crackling of salt thrown on the fire. They are possibly in some cases very fine mucous râles, but it is generally believed that they are due to abrupt opening up of collapsed alveoli whose walls have been stuck together by a small amount of exudation. These râles are heard in the early stage of pneumonia, in pulmonary œdema, and in collapse. Collapse explains their frequent occurrence at the posterior bases, and their disappearance after a few deep inspirations.

Pleural friction sounds are due to the rubbing of roughened pleural surfaces in respiration. Under normal conditions, the surfaces move on one another without noise, but if they are roughened by inflammatory exudation, tubercles or new growths, friction is produced. The sound may be soft or harsh. Sometimes it is very coarse and distinctly super-

ficial in quality. It may be heard at any part, but is specially common in the lower half of the posterior and lateral aspects of the chest. It is generally unilateral. It may accompany inspiration or expiration, or both, or a part of either. In many cases, the intrapulmonary sounds are unaltered. Coughing may remove râles or crepitations, but does not remove friction sounds.

Pleuro-pericardial friction (*pulsatile friction sound*) is a friction sound which is due to roughening of pleural surfaces, but its rhythm coincides with, or is at least modified by, the cardiac action. It is attributable to disease of that part of the pleura which overlaps the pericardium, and is accordingly heard about the margins of the superficial cardiac dulness. It may become less marked if the breathing is arrested, and examination may reveal undoubted pleural friction elsewhere.

THE SPUTUM.

In former centuries the sputum was studied with care, but it became somewhat neglected after the introduction of auscultation and percussion. In the latter part of last century, however, a new interest was aroused in its investigation, when the great significance of the presence of tubercle bacilli and elastic tissue came to be recognised.

In perfect health, there is little or no expectoration, any little secretion that comes up from the respiratory tract being caught by the pharyngeal muscles and swallowed. In large towns, however, where the atmosphere contains irritating substances, the normal secretions may be in excess, so that the individual has a slight spit. This consists chiefly of tenacious mucus, and often contains black carbonaceous pigment.

The *quantity* of the sputum in disease varies greatly. Children as a rule, and women sometimes, do not expectorate.[1] In pneumonia there may be scarcely enough

[1] In the case of little children, the sputum may be obtained for examination by covering the finger with gauze and inserting it into the aperture of the glottis. This excites cough, and the secretions of the respiratory tract are ejected on to the gauze.

in twenty-four hours to cover the bottom of the spittoon, while in bronchorrhœa it may amount to pints. In catarrhal affections, a diminution may take place before death.

The *odour* is usually disagreeable, but not strong. In bronchiectasis, and still more in pulmonary gangrene, it is horribly offensive.

The *colour* varies much. A mucous sputum is transparent and colourless, or may be blackened by carbon. The sputum may acquire various tints from the presence of blood (yellow, brown, red or green), or bile, or pigments (red, blue, etc.) employed in different commercial industries.

The *taste* is sweet when pus is present. Sometimes it is salt. In gangrene it is offensive.

The *consistence* varies much. In pulmonary œdema, it is watery. In pneumonia, it is very tenacious. The sputa may run together in the spittoon, or they may remain discrete as globular or 'nummular' masses. The latter are specially associated with phthisis.

The *specific gravity* also varies. Sputa that are thoroughly aerated float in water. Sputa which sink in water were regarded by the ancients as of bad omen ; they are suggestive of profuse suppuration, and therefore of phthisis, but they may be due to bronchitis.

The sputum may contain pieces of membrane in cases of diphtheria ; casts of the bronchi in fibrinous bronchitis ; moulds of the pulmonary alveoli in pneumonia ; calcareous particles from old tubercular foci in the lungs ; and Curschmann's spirals and Charcot-Leyden crystals in asthma.

A *mucous* expectoration (clear and tenacious) occurs in some healthy people in large towns, and is also seen in the early stage of acute bronchitis.

A *purulent* sputum may result from rupture of an abscess of the lung or liver, or of an empyema, into the air-passages.

A *muco-purulent* expectoration is the commonest of all. It occurs in acute bronchitis at any stage except the commencement. It is the variety regularly seen in chronic bronchitis.

A *serous* (watery) sputum is observed in œdema of the lungs, and sometimes in bronchorrhœa.

Blood is present in the sputum under many different circumstances. It is important to exclude blood derived from the gums, nose, etc. Blood in very small quantity may come from the larynx or trachea in bronchitis, as a result of the strain of coughing. It may also come from ulcerative lesions (tuberculosis, cancer, etc.) of the larynx, trachea and bronchi. Hæmoptysis, however, is usually due to some lesions in the lungs or finer bronchi.

In phthisis, there are two forms, the early and the late, the former being due to diapedesis or possibly to actual rupture of small vessels, and the latter to rupture of an aneurysm of a branch of the pulmonary artery. Hæmoptysis sometimes occurs in bronchiectasis, and in hæmorrhagic diseases such as purpura. In heart disease, hæmoptysis is a common consequence of the high tension in the pulmonary system of vessels. It is specially related to mitral disease, and particularly mitral stenosis. Hæmoptysis is also caused by pulmonary infarction, whether secondary to heart disease or to some other condition. Small leakages may take place from an aortic aneurysm into the trachea or into a bronchus for some time before the fatal hæmorrhage occurs. Vicarious menstruation, in the form of hæmoptysis, though occasionally genuine, is too often attributable to pulmonary tuberculosis.

The *prune-juice* expectoration seen in some cases of pneumonia going on to fatal œdema is dark-red or brown, watery, frothy and abundant.

Red-currant jelly expectoration is specially characteristic of malignant disease of the lung.

Elastic tissue in the sputum is an important evidence of phthisis. Quite exceptionally it is met with under other circumstances (bronchiectasis, gangrene, etc.).

Tubercle bacilli in the sputum are certain evidence of tuberculosis.

DISEASES OF THE NOSE AND NASO-PHARYNX.

i. Acute Coryza

(Acute Catarrhal Rhinitis and Naso-pharyngitis. Acute Nasal and Naso-pharyngeal Catarrh. Cold in the Head).

Etiology.—Acute coryza may result from the inhalation of irritating fumes, or from the internal use of certain drugs, such as iodide of potassium, or it may be part of an infectious fever such as measles. More frequently, however, it is an independent affection which is generally attributed to a chill. It often occurs in epidemics, especially in changeable weather. The immediate cause is probably a microbe, and a *Diplococcus coryzæ* or *Micrococcus catarrhalis* has been described.

Symptoms.—The patient feels out of sorts, sneezes, and has slight headache and chilliness. There may be slight elevation of temperature. The swelling of the mucous membrane and erectile tissue of the nose causes a feeling of dryness and obstruction, a sense of being 'stuffed up,' so that breathing is carried on through the mouth. There is a sense of dryness about the pharynx. The nasal condition causes impairment of smell, and the catarrh of the naso-pharynx is apt to involve the Eustachian tubes and interfere with hearing. Shortly after the feeling of dryness sets in in the nose, a thin serous exudation begins to flow in considerable quantity, and after a time this gives place to a highly purulent discharge. Still later the secretion becomes mucous and tough, and after a time it gradually ceases.

The disease usually runs its course in a few days, but the mucous discharge may go on for some time longer. The catarrh may spread up to the conjunctivæ and frontal sinuses, and down to the larynx, trachea and bronchi.

The important point in diagnosis is not to overlook such diseases as influenza and measles.

Treatment.—Exposure should be avoided. At bedtime the patient should have a hot mustard foot-bath, and a

diaphoretic powder or mixture. If there is fever, he should stay in bed. Great discomfort about the parts may be temporarily relieved by a spray of cocaine (1 per cent. solution of the hydrochloride). Menthol and camphor (of each 8 grains in vaselin 3 drachms) may be frequently applied to the nasal mucous membrane. When convalescence sets in, a bitter and acid tonic and a change of air may be recommended.

ii. Chronic Nasal Catarrh.

Three forms of this affection deserve notice :

(1) Simple Chronic Rhinitis (*chronic catarrhal rhinitis*, *chronic coryza*) is characterised by increased irritability of the nasal mucous membrane. The sufferer catches cold very readily, and the result is frequent temporary obstruction of the nose, with increase of secretion. Children who suffer thus have often postnasal adenoids. There is swelling of the mucosa with engorgement of the erectile tissue over the middle and lower turbinated bones. The sense of smell is not impaired. The disease sometimes progresses into the next form.

(2) Chronic Hypertrophic Rhinitis generally begins in childhood as a sequel of simple chronic rhinitis. It often arises in connection with postnasal adenoids or other conditions which obstruct the nares. The mucous membrane is red, and in various parts thickened from hyperplasia and granulation-tissue development. The lower turbinals, the septum and the naso-pharynx near the Eustachian tubes are the chief seats of hypertrophy. Mouth-breathing, deafness and a nasal tone of voice are apt to result.

The treatment consists in cleansing the nostrils by the frequent use of an antiseptic nasal spray ; removing adenoid vegetations, nasal spurs, etc., and applying special measures to the hypertrophied mucous membrane.

(3) Chronic Atrophic Rhinitis (*chronic fœtid rhinitis*, *fœtid coryza*, *ozæna*). Ozæna, though the term is sometimes applied to the disease now to be described, is, strictly speaking, a symptom (namely, the presence of fœtid nasal

discharge) which is observed in various diseases of the nose.

Chronic atrophic rhinitis is most common in girls, and usually begins in the second decade of life. The nasal mucous membrane, the erectile tissue and the subjacent bones undergo atrophy. The secretion dries into crusts, and a disgusting smell, due to the activity of the *Bacillus fœtidus ozænæ*, comes from the nose. The patient's sense of smell is lost.

The disease is incurable, but the fœtor may be minimised by frequent thorough cleansing. In debilitated subjects, cod-liver oil and tonics are indicated.

iii. Hay Fever

(Summer or Autumnal Catarrh. Rose Cold. Rose Fever).

Etiology.—Three elements may be recognised under this heading. First, there is a neurotic tendency, and this is probably why the disease may run in families. Secondly, there is a local predisposition in the nasal mucous membrane, which is often hyperæsthetic and presents the appearances of hypertrophic rhinitis. Thirdly, the immediate cause is something directly affecting the mucous membrane —sometimes changes of temperature, but very often pollen floating in the atmosphere. The disease does not confine itself to the nasal mucous membrane, but affects also the mucosa of the conjunctiva, mouth, pharynx, larynx and bronchi. Susceptible individuals suffer year after year, without acquiring any active immunity. The pollen of *Graminaceæ* is the principal cause of the hay fever of Europe and the 'June cold' of North America, whilst the pollen of certain species of *ambrosia* and *solidago*, as well as that of asters and chrysanthemums, is the cause of the more formidable and widespread autumnal catarrh of North America.

Symptoms.—Immediately after the pollen reaches the mucous membrane, itching sets in, and this is followed by the usual phenomena of coryza. Occasionally there is slight

fever. Paroxysms of asthma may occur in connection with the changes in the bronchial mucosa. The period of liability to hay fever usually extends over several weeks in summer.

Treatment.—Dunbar of Hamburg has prepared an antitoxic serum, which he terms 'pollantin,' by inoculating horses subcutaneously with a toxin obtained from active pollen. This toxin appears to be a toxalbumin, and though highly toxic for hay-fever patients, is harmless to non-predisposed individuals. Pollantin, when properly used, is curative in a large proportion of cases. It is not administered subcutaneously, but is applied to the surface of the affected mucous membrane. It is available in a liquid and a dry state. Some patients prefer the liquid for the eye and the powder for the nose. The dose of the liquid is one drop for each eye or nostril, and the corresponding quantity of the powder is about the bulk of a lentil. The applications should be made before rising each morning, and several times later on in the day. The doors and windows should be kept closed at night during the hay-fever season.

If pollantin is not available, or fails to give relief, the patient should remove for a time to a locality free from the exciting cause of the attack ; he might, for instance, take a voyage. To relieve the symptoms, cocaine solution (5 to 10 per cent.) or adrenalin chloride solution (1 in 1,000) may be carefully sprayed upon all the mucous surfaces involved. If the disturbance extends down to the bronchioles (*hay asthma*), the remedies employed in ordinary asthmatic attacks (stramonium, nitre paper, etc.) may be used. The local condition in the nose must be attended to ; the cautery may do much good in chronic hypertrophic rhinitis. It will often be advisable to give nervine tonics and sedatives, such as arsenic, quinine, valerianate of zinc and potassium bromide. A formula which has been recommended is a pill containing zinc phosphide (gr. $\frac{1}{5}$) and extract of belladonna (gr. $\frac{1}{4}$), to be taken thrice daily.

iv. Adenoid Vegetations

(Postnasal Vegetations. Adenoids. Hypertrophy of the Pharyngeal Tonsil).

These growths are due to hypertrophy of Luschka's tonsil (the pharyngeal tonsil), the mass of lymphoid tissue which extends across the roof and back of the naso-pharynx between the orifices of the two Eustachian tubes. Normally well marked in the young child, this tissue (like that of the faucial tonsils) tends to become firmer and less bulky after puberty. Hypertrophy to an extent that constitutes a postnasal growth is very common between five and fifteen, but may be met with at any age.[1] There may be no definite symptoms, and the growths may shrink or disappear of their own accord at puberty. But serious harm often results. The growths may obstruct the nares and cause mouth-breathing, with a consequent liability to frequently recurring catarrh of the nose, pharynx, larynx, and lungs. The growths and the catarrh lead to obstruction of the Eustachian tubes, deafness, and otitis media. Infections which specially attack the throat are rendered more dangerous by the presence of these growths. Deformities of the chest are brought about by the obstruction caused to inspiration. In bad cases, there is impaired mental development, characterised especially by inability to fix the attention (*aprosexia*). Adenoids may also give rise to various reflex neuroses such as asthma, incontinence of urine, sneezing, etc. Modern investigations show that chronic enlargement of the faucial tonsils almost never exists in children without corresponding disease of Luschka's tonsil ; and further, that adenoids are frequently present without enlargement of the faucial tonsils. The existence of postnasal growths can often be determined from the physiognomy. Corroborative evidence is obtained by digital exploration, and occasionally by posterior rhinoscopy.

[1] Though it was previously known to exist, the great frequency and importance of this disease were first pointed out by Meyer of Copenhagen in 1868.

Treatment.—As soon as any symptoms appear, the adenoids ought to be removed. In a large proportion of cases, tonsillotomy is indicated at the same time.

v. Epistaxis (Rhinorrhagia).

Etiology.—Bleeding from the nose occurs under many conditions, both local and general. It is specially prevalent between ten and twenty-five years of age, possibly owing to delicacy of the vessels, and also in elderly people whose vessels are likely to be degenerate. Among local causes are blows, picking of the nose, violent sneezing, fracture of the base of the skull, and catarrhal, strumous and syphilitic lesions. The general causes include hæmorrhagic diseases, such as hæmophilia, purpura and scurvy; the anæmias; cirrhosis of the liver; and certain infectious fevers, such as enteric. Epistaxis is common at puberty, and in the case of girls is sometimes regarded as vicarious menstruation. In elderly and plethoric persons it may be explained by the existence of high tension, and its occurrence may be followed by relief of headache, giddiness, and other premonitory symptoms of apoplexy.[1]

Morbid Anatomy.—The usual condition is oozing from veins or capillaries, due to increased blood-pressure, changes in the vessel-walls, or changes in the blood. Spontaneous epistaxis usually comes from the lower and anterior part of the cartilaginous septum; this is rarely dangerous, though apt to recur. In other cases bleeding may be from the anterior ethmoidal veins, which are closely connected with the intracranial circulation; such hæmorrhage may be profuse and obstinate. When due to general causes, such as hæmophilia and purpura, the bleeding may take place from numerous points of the mucous membrane.[2] In cases of trauma and ulceration the hæmorrhage may be arterial.

[1] Of epistaxis Sir Thomas Watson remarks: 'Sometimes it is a remedy; sometimes a warning; sometimes really in itself a disease' ('Lectures on the Principles and Practice of Physic,' 4th ed., 1857, i., 793).

[2] See A. Brown Kelly, *Lancet*, February 24, 1900.

If the cause is not at once apparent, the urine, heart, arteries, lungs and liver should be examined.

Treatment.—Slight attacks which tend to cease spontaneously do not call for treatment. In plethora or high tension, bleeding should not be hastily arrested. If the hæmorrhage is severe, cloths wet with ice-cold water may be applied to the nose and forehead as the patient lies on his back with the head slightly raised. Sometimes a douche of hot water acts better than cold. The arms should be elevated. Cotton-wool soaked with alum solution or with adrenalin chloride solution may be pushed into the nostril. If the bleeding-point can be discovered, it may be touched with the cautery. If these measures fail, the nares must be plugged.

DISEASES OF THE LARYNX.

i. Acute Catarrhal Laryngitis.

Etiology.—The disease is predisposed to by constitutional debility of any kind. The immediate cause is generally exposure to cold, but the catarrh may result from sudden changes of temperature, inhalation of irritant vapours or dust, and overuse of the voice. Of all causes, the inhalation of cold moist air by one who is habitually a mouth-breather, or whose nasal passages are temporarily obstructed by nasal catarrh, is perhaps the most important. The inflammation may be confined to the larynx, or may be part of a more extensive catarrh. It may occur by itself or be associated with some other disease, such as measles. It is most common in adults, but most dangerous in children.

Morbid Anatomy.—There is hyperæmia and swelling of the mucous membrane with disordered secretion. The latter is at first diminished in quantity, glairy, and rich in mucin. It afterwards becomes abundant and rich in degenerated cells.

Symptoms.—There is soreness and tickling about the larynx, with cough and hoarseness. Attempts to speak cause fatigue and sometimes pain. In severe cases, the voice is lost and the cough has a peculiar quality. If the

pharynx is simultaneously inflamed, deglutition may be painful. At the outset there are generally febrile symptoms. The symptoms usually pass off after a few days. Quite exceptionally the swelling is so great as to cause urgent dyspnœa, so that tracheotomy may be required to save the patient's life. The laryngoscope shows an increase of colour, but the vocal cords may be unchanged whilst other parts are much inflamed. Paresis of the cords is often observed, and is doubtless to a large extent mechanical. The disease as met with in children is separately described. (See *Spasmodic Laryngitis*.)

Diagnosis.—In doubtful cases this depends on laryngoscopic examination. The two diseases to be excluded are *diphtheria* and *laryngismus stridulus*. A history of exposure, and the presence of a more extensive catarrh of the respiratory tract, favour a diagnosis of laryngeal catarrh. In diphtheria the symptoms are likely to be more severe, membrane may be visible on the fauces and pharynx, and the lymph glands may be enlarged.

Treatment.—In severe cases the patient should rest in bed ; in mild cases he may sit in a warm room. A diuretic and diaphoretic mixture with a mercurial purge is often all the medicine that is necessary. For troublesome pain or cough, chlorodyne may be given at bedtime, and steam, medicated with chloroform, benzoin, or conium, may be recommended for inhalation. If the nose or naso-pharynx is at fault, menthol or some other topical application in the form of a spray or ointment should at once be prescribed. Any permanent cause of nasal obstruction must be attended to.

ii. Phlegmonous Laryngitis

(Œdema of the Larynx. Œdema Glottidis. Œdematous Laryngitis. Laryngeal Cellulitis).

Etiology.—Œdema of the larynx is much more frequently secondary than primary. It is often due to extension of inflammation from the pharynx or other neighbouring parts. It sometimes complicates syphilitic and tubercular laryngeal disease. It may, however, be due to mechanical

injury or to irritation by corrosive substances or boiling water. It may occur by itself as a result of septic infection, or may be associated with kidney disease or with various infectious diseases, such as scarlet fever, small-pox, erysipelas and pyæmia. Sometimes, however, it is primary, predisposed to by debility or fatigue, and excited by exposure. The disease is seldom seen in childhood.

Morbid Anatomy.—In the submucous tissue, there is an inflammatory exudation of a serous, sero-fibrinous or purulent character. The mucous membrane is swollen and dull red in colour. The ary-epiglottic folds and epiglottis tend to suffer most readily. In most cases the severe swelling is confined to the tissue covering one arytenoid, and as its bulk may be two or three times the normal, serious stenosis is produced.

Symptoms.—These often point to laryngitis of great severity, but the dyspnœa becomes urgent very quickly; and fatal suffocation may be the first sign that disease has existed anywhere. The diagnosis may be confirmed by the laryngoscope. The greatly swollen epiglottis may be felt by the finger, and may completely conceal the glottis from view. The condition is very dangerous. Under proper treatment, the attack may pass off after a few days, but the patient is not safe for some weeks.

Treatment.—The patient should suck ice, and an ice-bag should be applied over the larynx. The affected part should be promptly scarified with the laryngeal lancet. Pilocarpine should be injected hypodermically. If relief does not quickly follow, tracheotomy must be performed as low down as possible, care being taken that the knife actually passes through the swollen mucosa.

iii. Chronic Laryngitis

(Chronic Laryngeal Catarrh).

Etiology.—A preceding acute attack, persistent straining of the voice, and extension of a pharyngitis resulting from the inhalation of irritating substances, such as tobacco-smoke, are among the common causes. This condition and

pharyngitis together are often the consequence of mouth-breathing from nasal obstruction.

Morbid Anatomy.—There is hyperæmia of the mucous membrane, sometimes with slight swelling of the mucosa and dilatation of vessels. Small circular erosions are sometimes present on the cords and between the arytenoids.

Symptoms.—These include huskiness of the voice, tickling about the throat, and a dry cough.

Diagnosis.—A laryngoscopic examination is of great importance. In obstinate cases, the sputum and chest must be carefully examined for evidences of tubercle ; and in people who have reached middle life, the possibility of malignant disease must be borne in mind.

Prognosis.—Great improvement is often obtained by diligence in carrying out the proper treatment.

Treatment.—The causes of the disease must be avoided. Spices and other irritating articles of diet must also be avoided. The general health should be attended to, and a change of climate may be advisable. Locally, some astringent should be applied to the larynx from time to time—*e.g.*, solution of chloride of zinc (10 to 30 grains in 1 ounce of water). Nitrate of silver is of more doubtful value. Any unhealthy condition of the nose must be rectified.

iv. Laryngismus Stridulus (Child-crowing).

Etiology.—As in the case of the other convulsive disorders of childhood, rickets is the great predisposing cause, and the disease is accordingly promoted by poverty and defective hygiene. The irritation of teething, indigestion, and a fright are often possible causes. The disease is confined to the first few years of life, and is often associated with other affections of a convulsive character, such as carpo-pedal spasms or even general convulsions. It is a pure neurosis.

Symptoms.—The attack comes on suddenly, often during sleep. Respiration is arrested and the face becomes livid. There is tonic spasm of the adductor muscles of the larynx and often of the respiratory muscles, so that breathing is arrested and suffocation seems imminent. When the spasm

relaxes, the air rushing into the chest causes a crowing sound as it passes through the larynx. The spasm of the adductors is not always sufficient to prevent inspiration, and in such cases the attack may be more prolonged, and each inspiration is accompanied by the crowing sound. Attacks of spasm may recur many times daily, with or without obvious cause.

Prognosis.—In rare instances, death takes place in an attack, but recovery is the rule.

Treatment.—In the attack, cold water should be dashed on the face and chest. Inhalation of chloroform is an effectual measure. As long as there is any tendency to recurrence, bromide of potassium (dose for an infant, 3 grains) with or without chloral should be given. Once or twice a day the child should be placed in a warm bath, and have the upper part of the trunk sponged with cold water. It is important to give a prolonged course of cod-liver oil and chemical food in view of the constitutional condition, and the hygienic conditions must be carefully seen to.

v. Spasmodic Laryngitis

(Spasmodic Croup. False Croup. Spurious Croup. Stridulous Laryngitis).

These designations are applied to an affection of childhood which presents points of resemblance to acute laryngitis on the one hand and laryngismus stridulus on the other. It is to be regarded as a form of acute laryngitis with peculiarities which are due first to the small size of the larynx and secondly to the tendency to spasm at that age. The respiratory symptoms are severe, and may be the first evidence of the disease. The child is seized in the middle of the night with a violent barking or 'croupy' cough, with noisy inspiration or stridor, and with an aspect of threatened suffocation. The attack soon passes off, but may recur in succeeding nights. It is almost never fatal. During the day there may be slight cough and hoarseness, or no symptoms at all.

Treatment.—An emetic dose of ipecacuanha wine should be given, a poultice should be applied over the larynx, and

the child should be kept in an atmosphere of steam from a bronchitis-kettle. The bowels should be freely opened. Small doses of chloral or bromide may be given in the intervals, for a few nights, to ward off fresh seizures.

vi. Tumours of the Larynx.

Benign Tumours occur at all ages, but are most common in middle life. Males suffer more frequently than females. They are predisposed to by hyperæmia and catarrh of the larynx, and therefore the causes of catarrh, including the inhalation of irritating vapours, overstrain of the voice, nasal obstruction and syphilis, are also causes of new growths. The most common tumours in order of frequency are the papilloma, the fibroma, the polypus or fibrocellular tumour, and the cystoma. The myxoma, adenoma, lipoma, and angioma are uncommon. The vocal cords are the most frequent seat of growth.

Morbid Anatomy.—The *papilloma* often grows to the size of a pea, but is sometimes so large as to conceal the lumen of the glottis. It is sessile or pedunculated. The colour is pink, gray or white. The surface is warty. The tumour is often multiple. It (alone of the benign tumours) may recur after removal, though in other respects it is quite free from malignancy.

The *fibroma* (hard fibroma) is generally hard, sessile and smooth.

The *polypus* (soft fibroma, fibrocellular tumour) is usually small, reddish and translucent.

The *cystoma* is generally a retention cyst connected with the epiglottis.

Symptoms.—These vary greatly. The voice is almost always impaired. There may be cough. Respiration is sometimes embarrassed. Pain is exceptional. The general health is usually unaffected.

A laryngoscopic examination will permit a diagnosis to be made from chronic catarrh and infective new formations. Removal by operation is for the experienced laryngologist.

Malignant Tumours are usually squamous-celled epitheliomata, but sarcomata and alveolar carcinomata are occasionally observed. The growth may be extrinsic, spreading from the tonsil, tongue and pharynx ; or intrinsic, originating in the larynx itself. Intrinsic epithelioma commences with special frequency in the ventricle or on the upper surface of the vocal cord. Cancer is most common in the second half of life, whereas sarcoma occurs earlier.

Morbid Anatomy.—Commencing at the junction of the pharynx and larynx, epithelioma gives rise to congestion, thickening, and often ulceration of the mucous membrane. Commencing in the larynx, it constitutes an ill-defined, nodular growth, at first not unlike a simple tumour, but growing to a large size, and ulcerating at various parts of its surface.

Symptoms.—These will vary according to the place of commencement. A growth at the base of the tongue or in the pharynx will cause dysphagia. A growth near the glottis will impair the voice and afterwards embarrass respiration. Cough is not as a rule troublesome. The expectoration may contain blood and pieces of tumour tissue. Pain may for long be trifling or absent if the growth is intrinsic and unilateral ; but if it is extrinsic, or intrinsic and bilateral, there may be intense pain which radiates to the ear.

Diagnosis. — The age of the patient, the progressive development of symptoms, cachexia, the great and early deformity of the larynx, and the absence of the cicatricial changes of syphilitic disease are weighty evidence in favour of malignant disease.

Treatment.—The growth can occasionally be extirpated. In other cases, it is important to avoid all irritation of the affected region.

vii. Other Diseases of the Larynx.

Membranous Laryngitis is usually due to the diphtheria bacillus, but may result from the inhalation of irritating gases such as ammonia.

Syphilis and Tuberculosis of the larynx have been described in connection with these infections.

Perichondritis involving the cartilages of the larynx, and sometimes leading to necrosis of a part or the whole of a cartilage, may result from tubercular, syphilitic, enteric or cancerous ulceration. Traumata, and phlegmonous inflammations complicating acute fevers like typhus and erysipelas, are other causes. The inflammation does not necessarily cause death of the cartilage.

Symptoms.—The symptoms include pain and tenderness, difficulty in swallowing and breathing, impairment of voice, and cough. Swelling may be detected externally, or on laryngoscopic examination, according to the part involved.

Prognosis.—The prognosis is serious, not only owing to the risk of permanent interference with respiration and phonation, but also because suppuration may be followed by infection of the lungs, or may lead to death from exhaustion.

Treatment.—The treatment includes the local application of cold or of heat and the inhalation of medicated steam. If an abscess forms, it should be opened. If this cannot be done externally, a preliminary tracheotomy should be performed.

Paralyses of the Larynx are described in connection with disturbances of the cranial nerves.

DISEASES OF THE BRONCHI.

i. Acute Bronchitis

(Acute Bronchial Catarrh. Tracheo-Bronchitis. Cold in the Chest).

Etiology.—The causes are predisposing and exciting. With regard to *predisposing* influences, the young and the old suffer more than those in the prime of life. A sedentary life, and living or working in a hot and impure atmosphere, favour an attack. Constitutional debility, however induced; morbid blood-states as in Bright's disease and gout; heart disease; chronic bronchitis; a preceding acute attack; mouth-breathing, the consequence of nasal obstruction; and a moist, cold and variable climate are also predisposing influences.

Of the *exciting* causes, exposure to cold or wet, or to both together, is the most familiar, but it is probable that a sudden change from a cold to a heated and impure atmosphere can also induce the disease. Irritant vapours may cause it. It is sometimes an extension of catarrh from the naso-pharynx, and sometimes part of an infectious disease such as measles, whooping-cough, or influenza.

Numerous micro-organisms are found in the bronchial secretion, especially pneumococci and streptococci ; but how far these are to be regarded as the immediate causes of the disease is not yet known.

Morbid Anatomy.—The disease involves the larger and middle-sized tubes symmetrically. When the smallest tubes are inflamed, the condition is capillary bronchitis, which is described separately. The mucous membrane of the trachea and bronchi is red and swollen, and covered with an abundant secretion of mucus or muco-pus. The epithelium undergoes desquamation at the surface, while proliferating in its deeper layers.

Symptoms.—There is malaise with slight or moderate pyrexia, and the symptoms of coryza may be present. Unless in slight cases, breathing is accelerated. There is a sense of oppression in the chest, with rawness or actual pain in the region of the sternum. Cough worries the patient and causes pain in the chest ; often also in the forehead (congestive headache) ; sometimes in the epigastrium from strain of the abdominal muscles. It is at first almost dry, the expectoration consisting only of scanty, tough mucus (*sputum crudum*). After some days, however, the sputum becomes muco-purulent or purulent and abundant, and is easily brought up (*sputum coctum*). It may be slightly streaked with blood which comes from vessels in the mucosa of the upper air-passages, ruptured by the strain of coughing. Unless the chest has previously been disabled, there is little or no dyspnœa ; but if the individual has been subject to chronic bronchitis, an acute attack may give rise to severe dyspnœa and orthopnœa.

Physical Signs.—The principal physical signs at the height of the disease are dry râles and moist râles with normal per-

cussion. At an early stage, dry râles (sonorous and sibilant) may alone be audible ; later on moist râles may predominate, and in the stage of convalescence, a few moist râles may persist for a considerable time at the posterior bases. The dry râles may be associated with rhonchal fremitus.

In slight cases where the catarrh is chiefly in the trachea, there are often no abnormal physical signs.

Diagnosis.—This is usually quite simple. In *broncho-pneumonia* the dyspnœa is great, cyanosis and considerable fever are present, and there may be distinctive physical signs.

The question of *phthisis* arises chiefly in connection with chronic bronchitis, but acute phthisis sometimes presents a resemblance to acute bronchitis. Moist râles of a clicking or consonating quality suggest tubercle. Moreover, moist râles tend to predominate at the bases in bronchitis and at the apices in phthisis. The sputum should be examined for tubercle bacilli and elastic tissue. If high fever persists for any length of time, ordinary bronchitis may be excluded.

It is important to bear in mind the possibility of an infectious or other disease, of which the bronchitis may be a part or a complication.

Prognosis.—In a previously healthy adult, convalescence usually sets in within a week or ten days, and there is no danger to life. But in a patient who has a serious degree of emphysema, or who is suffering from heart disease, an attack of acute bronchitis may cause death through the strain upon the right ventricle. In children, especially after measles and whooping-cough, the catarrh is apt to spread to the finest tubes and give rise to broncho-pneumonia, and a similar danger is present in the case of old and feeble persons.

Treatment.—The patient should take a hot mustard foot-bath and a hot drink, and go immediately to a warm bed. A dose of chlorodyne (10 minims for an adult) or Dover's powder (10 grains) may be given the first night to relieve the malaise, the soreness in the chest and the dry cough. A simple diaphoretic mixture containing a depressant expectorant such as ipecacuanha or antimony should thereafter be given for a day or two, and the bowels should be

gently opened. When the skin becomes moist, stimulant expectorants are indicated, such as ammonium carbonate, spirit of chloroform and squill. When convalescence is well established, an acid tonic with nux vomica may be given, and the patient should have a change of air to complete the cure.

If there is much moisture in the chest, or if there is cyanosis, and especially in the old and young, opiates must be avoided. Under such circumstances, it is a good plan to give children an emetic dose of ipecacuanha wine, while adults may (as recommended by Wm. Murray) inhale the vapour from a mixture of 1 ounce of turpentine and 4 ounces of boiling water in a Maw's inhaler.

ii. Capillary Bronchitis

(Suffocative Catarrh. Peripneumonia notha).

This disease is observed chiefly in childhood and advanced life. It may be due to extension from the larger tubes, or may occur independently. In children, it is specially apt to follow measles and whooping-cough, and to become associated with pulmonary changes. The purulent secretion, by blocking the bronchioles, leads to collapse of the corresponding lobules of the lungs. From the clinical point of view, capillary bronchitis is practically the same thing as broncho-pneumonia, and this will be considered in connection with diseases of the lungs.

iii. Chronic Bronchitis

(Chronic Bronchial Catarrh. Winter-cough).

Etiology.—Chronic bronchitis is most common in elderly people. In many people it recurs with every return of the cold and wet season, to disappear more or less completely in summer. It may result from repeated attacks of the acute disease. Heart disease, chronic lung disease, kidney disease, gout, and the inhalation of irritant substances are other causes.

Morbid Anatomy.—The condition of the bronchial mucous membrane varies greatly; it may be either thickened or

thinned. Emphysema is always present. The right ventricle of the heart is likely to be hypertrophied.

Symptoms.—Cough and spit are the most constant symptoms. If the disease has lasted long, there is shortness of breath on exertion, but this is attributable to the coexisting emphysema. There is no fever, and the general health may be very good. Hypertrophic osteo-arthropathy is a rare complication.

Physical Signs.—The physical signs very often include those of emphysema (diminution of superficial cardiac dulness, enfeebled respiratory murmur, prolongation of expiratory murmur, percussion note resonant or hyper-resonant) ; and in addition there may be dry râles and at the posterior bases a few moist râles. The sputum is usually muco-purulent and frothy. Sometimes it is very abundant (*bronchorrhœa* or *chronic pituitous catarrh*). Sometimes there is little or none, though the cough may be very troublesome (*dry catarrh* or *dry bronchitis*). Fœtid expectoration (*fœtid* or *putrid bronchitis*) suggests decomposition of secretions, as in bronchiectatic and tubercular cavities ; or of tissues, as in pulmonary gangrene.

Diagnosis.—The principal difficulty is in distinguishing between certain cases of chronic bronchitis and chronic phthisis. The symptoms and physical signs may be common to the two diseases, and it is probable that many of the cases recognised after death as instances of ' healed phthisis ' have been regarded while the pulmonary ailment was active as bronchitis. Moreover, winter-cough may pass into phthisis. The most important distinction lies in the presence or persistent absence of the tubercle bacillus. Hectic fever going on for weeks, and hæmoptysis, are of course in favour of phthisis ; but their absence does not exclude it. Similarly the absence of physical signs of phthisis does not exclude tuberculosis.

Prognosis.—Much depends on the social circumstances of the patient. In children who are well cared for, the disease may be got rid of in the course of some years. Elderly people who suffer only in winter may, by going abroad, escape fresh attacks, and thus become practically cured,

unless much emphysema is already present. The prognosis has to be modified if the cause is some incurable disease, affecting, for example, the heart, lungs, or kidneys. In elderly people there is the risk of an acute exacerbation, perhaps with fatal extension to the capillary bronchi. In the majority of hospital patients, there is the prospect of acute or subacute attacks every winter, with increasing emphysema and disablement of the right heart.

Treatment.—It is important to attend to, and if possible rectify, any gouty condition or any disease of the heart, liver, kidneys, or other viscera. An occasional mercurial laxative may be given with advantage. The clothing must be warm, with flannel always next the skin, but it should not be oppressive. Patients must try to avoid getting fresh colds when the winter sets in. They should winter, if possible, in a mild and dry climate.

Of all medicines, cod-liver oil is most to be recommended. It should be taken all through the winter, and if it be begun before the cold season commences, it may help to ward off a fresh attack. Stimulating liniments may be rubbed into the chest to cause moderate counter-irritation. If the spit is tough and difficult to get up, an alkali (*e.g.*, potassium iodide in 5 or 10 grain doses) is indicated. If it is abundant and easily brought up, an acid should be given, with nux vomica and perhaps senega, with a view to diminish the quantity. Creosote or one or other of the volatile oils (turpentine, clove, eucalyptus, etc.) may be inhaled if the sputum is fœtid. If there is much irritability of the mucous membrane, as indicated by severe coughing with little or no expectoration, it may be advisable to give an opiate in small doses at bedtime.

iv. Membranous Bronchitis

(Plastic, Fibrinous, or Croupous Bronchitis).

Etiology.—The disease is rare and its etiology is quite obscure, but a considerable proportion of the sufferers are tubercular. It may occur at any age, and has been observed

in different members of a family. It is more common in males than in females.

Morbid Anatomy.—The branching casts which are coughed up are white, fibrinous-looking moulds of a smaller or larger portion of the bronchial system of tubes. They are mostly derived from the smaller tubes. The structure of a cast is in concentric layers, and in its midst there may be leucocytes and epithelium. While many casts appear to consist of fibrin, there are others in which this cannot be found, but which are rich in mucin. The bronchial mucous membrane may be congested or pale. Its epithelial lining may be preserved or may have disappeared.

Symptoms.—Two varieties of the disease are described, namely, *acute* and *chronic*. The former is generally associated with some infectious fever. It resembles bronchitis, but is differentiated by increasing dyspnœa and the expectoration of casts. In the chronic variety, there are paroxysms which recur at regular or irregular intervals during many years. The patient suffers from oppression or pain in the chest, cough, and dyspnœa. After a period of hours or days, the symptoms become urgent, and then the cast is ejected, usually along with sputum of a more ordinary kind, and sometimes with blood. If the cast is brought up in small fragments, the nature of the case may for a time be overlooked. Slight pyrexia may accompany the attack.

Physical Signs.—These are not very definite. There may be enfeeblement or loss of the breath-sounds over those portions of lung whose bronchial tubes are obstructed. These portions of lung may become collapsed or inflamed, so that dulness is detected on percussion.

Diagnosis.—This depends on the recognition of the fibrinous-looking casts, though the symptoms and the history of the case may be suggestive. Diphtheritic moulds of the trachea and bronchi must not be mistaken for these casts.

Prognosis.—The acute form is sometimes fatal from asphyxia. The chronic variety is not dangerous unless some complication be present, and ultimate recovery may be hoped for, though possibly only after many years.

Treatment.—The inhalation of atomised lime-water has

been recommended, and also the intratracheal injection of olive oil. An emetic may help to expel the membrane. Pilocarpine has also been suggested, and iodide of potassium may be given internally.

v. Bronchiectasis (Dilatation of Bronchi).

Etiology.—Apart from very rare congenital cases, bronchiectasis, or dilatation of bronchial tubes, occurs only in connection with some other lesion in the bronchi or lungs. One very important factor in its production is weakening of the bronchial wall, and another is increase of the distending force within. Chronic catarrh leads to softening, atrophy, and weakness of the wall. Obliteration of fine bronchi will allow the inspired air to exert greater distending pressure on the larger tubes with which those fine bronchi are connected. And again, obstruction of a bronchus (*e.g.*, by syphilitic stenosis, aneurysm, or malignant growths) will cause the secretions to accumulate, and thus lead to dilatation behind the obstruction.

Morbid Anatomy.—Permanent dilatations of the bronchial tubes may be classed as *cylindrical* or *sacculated*. The two kinds may be present together. The former variety occurs chiefly where the cause has been a general one, as in chronic bronchitis. The sacculated form is suggestive rather of a local process, and is best seen in fibroid phthisis, where it is induced not only by the mechanisms already mentioned, but also by direct traction of the hypertrophied connective tissue, which passess outwards to the adherent pleura. A bronchiectatic cavity is continuous with a bronchus, and though the wall is thinned, there is still a distinct lining membrane. Decomposition of the muco-purulent or purulent contents may lead to ulceration of the wall, and at the same time render the sputum highly fœtid, so that it resembles that of gangrene.

Symptoms.—Most of these are referable to the chronic bronchitis, fibroid phthisis, or other primary disease. But if a large saccular bronchiectasis is present, in which the secretions may collect, a very characteristic symptom may be noted, viz., cough occurring from time to time in

paroxysms, which are associated with profuse expectoration. A paroxysm can sometimes be induced by a change of posture. In course of time the sputum becomes fœtid—another important evidence of the disease.

The fœtid sputum on standing settles into three layers, the uppermost of which is frothy, the middle one watery and comparatively clear, and the lowest dense and purulent. Among the pus in the bottom layer there may be recognised Dittrich's or Traube's plugs, which are yellowish masses varying in size up to that of a bean, and consisting of pus, bacteria, fatty acids, etc. If ulceration has taken place, elastic tissue may be present in the sputum.

Dyspnœa and lividity on exertion, clubbing of the digits, and hæmoptysis are not uncommon.

The **physical signs** are those of the primary disease, but a large sacculated bronchiectasis near the surface will yield the usual signs of cavity (cavernous breathing, etc.).

Abscess of the brain and hypertrophic osteo-arthropathy are occasional complications.

Diagnosis.—Tubercular cavities are generally situated at the apex ; bronchiectatic cavities lower down. In phthisis, the signs of consolidation precede those of cavity, while the converse is true of bronchiectasis. The temperature is higher in phthisis with cavities, and the disease is progressive, whereas it may long remain stationary in bronchiectasis. Tubercle bacilli in the sputum point to phthisis, while the paroxysmal cough and abundant fœtid sputum suggest bronchiectasis. When the sputum is very offensive, the history of the case, mode of onset and accompanying symptoms are of importance in the diagnosis from gangrene.

Prognosis.—Cure cannot be expected, but the patient may for years enjoy fair health.

Treatment.—The primary disease should be attended to. Cod-liver oil and creosote or guaiacol carbonate should be given internally. When the breath is fœtid, frequent inhalations of creosote or some other antiseptic should be employed. Intratracheal injections have been recommended (*e.g.*, either menthol, 10 parts ; guaiacol, 2 parts ; olive oil, 88 parts—1 drachm twice a day ; or izal in glycerin,

10 per cent.). They are easily administered with a suitable syringe, without the aid of the laryngoscope. The creosote chamber is a useful agent. The patient is put in a small room, where a drachm of creosote is poured upon water in a saucer, and caused to evaporate by placing a spirit-lamp under the saucer. This treatment should be carried out for a quarter of an hour every second day to begin with, but eventually for an hour every day. If the bronchiectatic cavity can be accurately localised, it may be opened through the chest-wall and treated on ordinary surgical principles.

vi. Spasmodic Asthma (Bronchial Asthma).

Etiology.—The disease is most common in males. It may occur at any age. The tendency is frequently inherited. One of the important causes is the condition of the atmosphere, but while many sufferers blame dampness, and many others closeness of the atmosphere, a great deal depends upon individual peculiarity, and it is well known that some asthmatics who suffer much in the country may enjoy good health in a large town. The most common cause is bronchial catarrh, which may leave the mucosa in an abnormally irritable state. Or an enlarged bronchial gland in cases of bronchitis or phthisis may compress the vagus or some other nerve in the chest. Morbid blood-states may be causes, as in gout and kidney disease. Nasal polypi and other nasal affections, and the causes of hay fever, may be causes of asthma.

An attack may be excited by emotion, the inhalation of various kinds of dust, the odour of particular plants or animals, certain articles of diet, or a loaded stomach.

Morbid Anatomy.—This is unknown, as death does not take place during an attack. In cases of old-standing, bronchitis and emphysema are present.

Pathology.—The characteristic symptoms are generally held to be due wholly or in part to spasm of the muscular wall of the bronchial tubes. Another theory is that the obstruction in the tubes is due to hyperæmia and swelling of the mucosa with secretion of tough mucus. Some, again,

would explain the attack by spasm of the diaphragm, or by reflex spasm of all the inspiratory muscles, or by bronchiolitis, or by irritation of the mucosa by the pointed Charcot-Leyden crystals which are found in the sputum. In any case, it may be said that the asthmatic paroxysm is in great measure an expression of a neurosis; while the paroxysm itself may be excited in various ways, including direct irritation of the bronchial mucosa and reflex processes set in operation by irritation of other mucous surfaces.

Symptoms.—In some cases there are premonitory symptoms (*aura asthmatica*), such as lassitude or a sense of oppression in the chest. Often, however, the paroxysm is quite sudden in its onset, and a common time for its commencement is about 2 a.m. The patient is awakened by a sense of suffocation which compels him to sit up, and he sometimes leans over the open window to get as much air as possible. The chest is fixed in the position of deep inspiration, and in the effort to get breath all the muscles of forced inspiration are brought into play. The frequency of respiration is not much increased. Expiration is prolonged, and associated with wheezing. The pulse is feeble, the face pale and anxious-looking. The temperature is normal or subnormal. There is a great increase of the eosinophile leucocytes in the blood.

Abundant dry râles are heard over the chest, masking the normal respiratory sounds. Percussion is normal or hyperresonant. The sputum contains Curschmann's spirals and Charcot-Leyden crystals. Curschmann's spirals are spirally wound casts of the bronchioles, consisting of mucin and cells; some of them possess a well-marked central core. They have been regarded as due to acute bronchiolitis. They occur early in the attack, and appear in the sputum in the form of little balls not unlike sago grains. When unwound on a glass they may reach 1 inch in length. Charcot-Leyden crystals ('asthma crystals') are colourless, pointed octahedra, which consist of an organic phosphate. (They are found also in the blood in leucocythæmia.)

Attacks of asthma may last for minutes, hours or days. They may occur night after night.

Diagnosis.—This is usually easy, but it should be remembered that a certain amount of spasmodic dyspnœa may accompany acute bronchitis and cardiac and renal disease. When the obstruction is in the larynx, there is laryngeal stridor, and the dyspnœa is inspiratory, whereas in asthma the dyspnœa is mainly expiratory. The dyspnœa of emphysema is permanent and not purely spasmodic. Aortic aneurysm and other mediastinal tumours, which may induce asthmatic attacks by pressing upon nerves, are often associated with other pressure-symptoms, as well as with distinctive physical signs.

Prognosis.—This is hopeful in early life if the cause can be removed. After middle life, and especially if chronic bronchitis, emphysema, or any other irremovable cause is present, recovery cannot be expected.

Treatment.—Relief may be given in the attack by inhalation of chloroform, iodide of ethyl, or nitrite of amyl ; by hypodermic injection of morphine or of pilocarpine ; by a hot drink containing whisky ; or by inhalation of the smoke evolved by burning plants like stramonium, tobacco, and lobelia, or by burning nitre paper (blotting-paper soaked in strong solution of nitre and dried). An emetic, a mercurial purge, and the compressed air-chamber are other remedies which deserve mention.

In the intervals, iodide of potassium, arsenic, bromide of potassium and belladonna should be given, either in combination, or separately, in the order in which they are named. The patient should be careful as to his diet, and must avoid heavy meals near bedtime. The bowels must be kept regular. Changes of climate must at first be experimental, since individual idiosyncrasy is so potent in this disease ; but, speaking generally, the large towns with their smoke and fog are more likely to suit than the purer atmosphere of the country.

vii. Obstruction of the Trachea and Larger Bronchi.

Obstruction of the larger air-tubes may be due to (1) a foreign body in their lumen ; (2) stricture from disease in their wall ; and (3) narrowing by compression from without.

Obstruction of the Trachea.

(1) A *foreign body* in the trachea may be large enough to cause complete obstruction and lead to immediate death. If it is smaller it causes dyspnœa with paroxysmal exacerbations. A body such as a pin may cause pain without dyspnœa. In rare instances there are no symptoms.

As a rule, however, a foreign body is not retained in the trachea, but passes into a bronchus—most commonly the right, whose orifice is larger, and occupies more of the lower end of the lumen of the trachea, than that of the left. Tracheotomy is generally necessary for the removal of a foreign body from the trachea.

(2) *Stricture* of the trachea is almost always due to syphilis, though in rare instances it may be due to growths originating in the wall of the tube, or to leprosy, tuberculosis, or some other infection. The stricture is sometimes annular and of limited extent, but more frequently it extends for some distance along the lower part of the trachea, and it may involve a portion of the bronchi. The symptoms include dyspnœa and stridor, the latter being often expiratory as well as inspiratory. The larynx is not moved extensively up and down as in laryngeal obstruction, and the laryngoscope reveals no evidence of laryngeal disease. It is important to exclude all possible causes of compression. A history of syphilis and other signs of that infection are valuable corroborative evidence.

Treatment generally includes tracheotomy. This is done below the obstruction if practicable. If the obstruction is too low down for this, a flexible tube should be passed from the tracheotomy wound through the stricture. A course of potassium iodide should be inaugurated as soon as the diagnosis is made.

(3) *Compression* of the trachea may be caused by goitres and other tumours in the neck, aneurysms and other tumours in the thorax, diseased bronchial glands, and mediastinal abscesses. In many of these cases the cause of compression is quite obvious. As in stricture, dyspnœa and stridor are important symptoms. The paroxysmal character

of the dyspnœa is sometimes attributable to accumulation of secretion below the obstruction.

The treatment must obviously be directed to the cause of compression. An enlarged thyroid may be partly excised. If the compression is at the upper part of the trachea, tracheotomy may be indicated.

Obstruction of the Larger Bronchi.

(1) A *foreign body* in a bronchus may become impacted and cause permanent obstruction. Ulceration of the mucosa may ensue, with sloughing and hæmorrhage. Bronchiectasis and abscess may follow. The secretions which accumulate behind the obstruction may decompose, and by insufflation give rise to diffuse gangrene. The situation of the foreign body may possibly be ascertained by physical examination and skiagraphy, and removal is then for the surgeon to attempt.

(2) *Stricture* of a bronchus or of both bronchi is caused by syphilitic cicatricial processes which may at the same time involve the trachea. Dyspnœa is a common symptom. An important combination of physical signs consists in normal percussion with enfeeblement or absence of the respiratory murmur on the affected side of the chest. Antisyphilitic treatment is indicated, but can scarcely be expected to bring about a cure.

(3) *Compression* of a bronchus is produced by aneurysms, diseased bronchial glands, and other intrathoracic tumours. An aneurysm of the lower aspect of the aortic arch will readily compress the left bronchus which passes under it, and will at the same time give rise to the important symptom known as *tracheal tugging*. The results of compression include dyspnœa and enfeeblement or loss of the respiratory murmur with preservation of clear percussion. Complete obstruction of a main bronchus may, however, lead to collapse of the lung, with dulness on percussion; loss of respiratory murmur, of vocal resonance and of vocal fremitus; and shrinking of the chest on the affected side. Treatment must obviously be directed to the cause of the compression. Otherwise it is purely symptomatic.

DISEASES OF THE LUNGS.

i. Congestion of the Lungs.

Active congestion is present in the early stage of inflammation, and under other circumstances, but is not of clinical importance.

Passive hyperæmia is of two kinds : (1) Mechanical congestion, and (2) hypostatic congestion.

Mechanical Congestion results from mitral regurgitation and even more readily from mitral obstruction. The mitral lesion leads to excessive accumulation of blood in the left auricle and pulmonary veins, and the resulting hypertrophy of the right ventricle keeps up an abnormal pressure in the pulmonary circulation. The capillaries become dilated and tortuous, the connective tissue is increased, and hæmorrhages take place into the alveoli, and also into the connective tissue, which thus becomes pigmented. The lungs are brown in colour and denser than normally (*brown induration*). There is catarrh of the bronchial mucous membrane, and the larger pulmonary arteries are sometimes atheromatous.

The principal symptoms are dyspnœa, cough and palpitation ; but, apart from exertion, these may be absent so long as compensation remains good. Hæmoptysis is common. Physical signs of a mitral lesion are likely to be recognisable. Fine moist râles may be heard over the lungs. Such patients are apt to suffer from frequent attacks of bronchitis. The prognosis depends upon the cardiac condition, and treatment must be directed chiefly towards the heart.

Hypostatic Congestion involves the dependent parts of the lungs, and is therefore most commonly found at the posterior bases. It results from great weakness of the heart, as in typhus, enteric and other prolonged and severe debilitating conditions. The affected portions of lung are œdematous and engorged with dark blood. Sometimes a stage further than congestion is reached, and portions of lung become consolidated by a low form of inflammation, so that the alveoli do not contain air, and the tissue sinks in water (*hypostatic pneumonia, splenisation*).

Symptoms.—When hypostatic congestion is present, the patient suffers from cyanosis, rapid and shallow breathing, and of course great prostration.

Physical Signs.—Over the affected portions of lung there are impairment of percussion, enfeeblement of the respiratory murmur, and moist râles.

Treatment.—The preventive treatment of this condition is very important. The patient's posture should be changed from time to time ; he should be carefully fed ; and stimulants and tonics, including ammonia, strychnine, quinine, and perhaps strophanthus or digitalis, are indicated. Oxygen inhalations should be tried if lividity develops, and large poultices of linseed-meal should be frequently applied over the affected areas.

ii. Œdema of the Lungs.

The serous fluid is present in the pulmonary tissues and also in the alveoli. Œdema is due largely to the same class of conditions as give rise to passive congestion ; and in addition, it is promoted by morbid blood-states which tend to be associated with dropsy elsewhere—*e.g.*, Bright's disease and anæmia. The dependent parts of the lungs suffer most as a rule, but a localised œdema may be found at times far removed from the base. The affected portion of lung is bulky and wet, but floats in water. On section, much watery and frothy fluid exudes. Very commonly, after death from almost any disease, congestion and œdema are found together in the lower portions of the lungs ; their presence is often doubtless due to the mode of death.

Symptoms.—The patient suffers from dyspnœa and cough with an abundant watery (serous) and frothy expectoration ; and in addition, there are the symptoms of the primary disease.

Physical Signs.—Percussion is impaired over the affected parts, the respiratory murmur and vocal fremitus are enfeebled, and fine moist râles are audible. It must be remembered, however, that impairment of percussion in such cases may be due to pleural effusion.

The prognosis depends mainly upon, and treatment must

be directed to, the original affection. Dry cups and mustard poultices may be recommended as appropriate local measures.

Acute Suffocative Pulmonary Œdema.

This somewhat rare condition is characterised by the acute or almost sudden onset of great dyspnœa or orthopnœa, with the expectoration of an abundant watery and frothy sputum. The pulse is accelerated, the surface may be blue or pale, the extremities cold, and the distress or sense of oppression great. Râles are heard over the chest. The patient may have been in the midst of apparent health, though probably suffering from some organic disease, either acute or chronic, in the abdomen, chest, or head. The sputum is brought up with very little effort, and has a characteristic appearance. While it may be white to begin with, it soon acquires a pink colour, this tint being specially well shown by the frothy portion. It may be actually bloody in appearance. The duration of the attack is from half an hour to twelve hours, and in the course of that time a couple of pints of sputum may be put up. A patient may pass through several attacks of the kind in a period of months or years, but sometimes the first attack proves fatal. The condition appears to be met with especially in women in middle life, not infrequently in the evening or at night, sometimes after a hard day's work, and occasionally after coitus.

This dangerous form of general œdema of the lungs is probably due to relative failure of the left ventricle, while the right ventricle continues to act strongly, thus leading to an accumulation of blood at high pressure in the pulmonary veins. When the left ventricle recovers itself, recovery from the symptoms may take place quickly. A less satisfactory explanation that has been suggested is that it is due to increased permeability of the walls of the pulmonary capillaries and alveoli.

Inhalations of chloroform and hypodermic injections of morphine have both proved to be highly beneficial in the treatment of the paroxysm. If cyanosis is very marked,

blood-letting may be advisable. In addition to these measures, it may sometimes be desirable to give stimulants by the mouth, by the rectum, or hypodermically. Heat should be applied to the extremities, and sinapisms to the seat of oppression in the chest.

iii. Obstruction of the Pulmonary Artery.

Obstruction of a branch of the pulmonary artery is usually due to embolism, and this usually has its source in the right side of a dilated heart. Much less commonly it is due to thrombosis in the artery itself, a condition which occurs chiefly in mitral disease with obstruction to the pulmonary circulation. As a result of obstruction, a *hæmorrhagic infarction* develops in many cases, though not in all. The large size of the pulmonary capillaries and the presence of the bronchial artery help to save the lung. The infarct is firm, dark and wedge-shaped or conical, with its base on the pleura, which becomes covered with fibrin. The alveoli, the fine bronchial tubes and the capillaries at the seat of lesion are engorged with blood. The condition is sometimes known as 'pulmonary apoplexy,' but it is seldom that the lung tissue is torn so that a cavity is produced as in cerebral apoplexy. It is believed that an infarction sometimes clears up altogether. Sometimes it becomes fibrous and shrunken. Sometimes it dies and is replaced by a cavity. Sometimes the dead part becomes gangrenous.

Symptoms.—The symptoms of pulmonary infarction are not always definite. Sometimes, however, the onset of a severe stitch in the side, with hæmoptysis, pleural friction-sound, and hollow respiratory murmur at the seat of pain, in a patient who is disabled by chronic heart disease, makes the condition of matters quite evident.

Occasionally the displacement of a thrombus from the veins of the thigh, pelvis or uterus after parturition, or from some other situation, leads to obstruction of the main trunk or a primary branch of the pulmonary artery. Under such circumstances, death may take place immediately, or after a few minutes of orthopnœa, lividity and terror. Where a less important vessel is plugged, symptoms of a similar

kind, but of less violence, may be present for half an hour or so and then subside. Where death takes place suddenly from obstruction of a main trunk, there is no infarction.

Other kinds of pulmonary embolism require only to be mentioned here. *Septic embolisms* followed by multiple abscesses are a feature of many cases of pyæmia. *Fat embolism* may occur after fractures of bone, but is seldom very serious. *Air embolism* may occur in the course of surgical operations which involve wounds of large veins. In these cases there is danger to life through the mixture of blood and air being churned into a froth by and in the right ventricle, so that the blood does not get into the pulmonary arteries, and the circulation is arrested.

iv. Collapse of the Lungs and Atelectasis.

Atelectasis is congenital, while collapse is an acquired condition. In the former, a portion of one or both lungs remains unexpanded as in the fœtus, whereas collapse is a reversion to the fœtal condition after expansion has taken place.

Collapse (Apneumatosis).

Etiology.—The yielding character of the chest-wall in children, and particularly in rickety children, is favourable to collapse. Weakness of the respiratory muscles—*e.g.*, in severe febrile conditions—is another factor. Pressure on the lung by liquid or gaseous accumulations in the pleural cavity is a common cause. Another is obstruction of the respiratory tract, which may be due to compression from without, *e.g.*, by a tumour, or to something in the wall or in the lumen of the air-passages, such as adenoids, foreign bodies, or inflammatory secretions. The obstruction may act like a ball-valve, and actually exhaust the air out of the pulmonary alveoli ; or it may simply prevent the movement of the air either outwards or inwards, with the result that the imprisoned air undergoes absorption and the vesicles collapse.

Morbid Anatomy.—The collapsed lung or portion of lung is reduced in size. The condition is often limited to several

individual lobules or groups of lobules. Collapsed portions at the pleural surface are depressed. The affected foci may be scattered irregularly throughout the lung, or may be chiefly at the base. They are bluish-gray in colour, dry, smooth, firm and non-crepitant, and sink in water. A lung in this state is described as *carnified*, and it ultimately becomes leathery. Various other anatomical features will result from the cause of the collapse.

Symptoms. — These naturally vary according to the primary disease. Of those which are more definitely referable to the collapse, the most important are dyspnœa, cough, cyanosis, a rapid pulse, restlessness, and prostration.

Physical Signs.—Besides those which depend upon the primary condition, there may be retraction of the chest-wall, especially at its lower part, and inspiratory retraction of the intercostal spaces. If the collapse is extensive, there may be impairment of percussion, enfeeblement of respiratory murmur, and bronchial breathing.

Diagnosis.—This depends upon the presence of a disease which is liable or certain to be associated with collapse, and on the development in the course of that disease of the symptoms and physical signs just indicated.

Prognosis.—Extensive collapse occurring in the course of bronchitis, measles or whooping-cough is very serious, and is apt to lead to death by asphyxia. In slighter cases, if the child is naturally vigorous and is well cared for, the prognosis is favourable.

Treatment.—Careful nursing and feeding, abundance of fresh air, and often stimulation, are necessary. Any respiratory obstruction must be removed if possible. Tracheotomy may be required for laryngeal diphtheria. An emetic (ipecacuanha or sulphate of zinc) is valuable if the secretions are blocking the bronchial tubes.

Atelectasis.

This condition has been attributed to various causes, such as enlargement of bronchial glands, but is doubtless due chiefly to feebleness of the child and inability of the respiratory muscles to expand the chest. The anatomical condition

is similar to that in collapse, but as there is no primary disease, the anatomical condition, symptoms, and physical signs are simpler than those often present in collapse. General feebleness, lividity, rapid and shallow breathing, and inspiratory retraction of the lower part of the chest are observable. The respiratory murmur is feeble, and there may be some impairment of percussion at the bases.

Treatment.—A warm atmosphere, hot baths, gentle friction of the surface, and sometimes artificial respiration, are the measures indicated.

v. Broncho-pneumonia

(Lobular or Catarrhal Pneumonia).

Etiology.—Broncho-pneumonia is most common in the first five years of life, but is also common in old people. It is sometimes primary, but more often secondary. It often follows or complicates the specific fevers, especially measles, whooping-cough, and diphtheria. It is sometimes due to extension of catarrh from the larger bronchial tubes. It occurs in those who are enfeebled by age, by paralysis, or by any debilitating condition. It may be excited by the entrance into the air-passages of food, drink, or discharges from sores or wounds about the mouth, nose, larynx, or pharynx (*aspiration-pneumonia*). In septic cases the inflammation may go on to suppuration and gangrene. Broncho-pneumonia occasionally follows the inhalation of ether for anæsthetic purposes. In two cases which came under my care, the inhalation of gaseous ammonia produced a membranous inflammation of the respiratory tract with broncho-pneumonia. In one of them, the fibrinous exudation extended from the larynx to the finest tubes.[1] Among predisposing causes of broncho-pneumonia may be mentioned deficiency of food, impure air, rickets, and diarrhœa. The specific broncho-pneumonia due to the tubercle bacillus is separately considered under Pulmonary Tuberculosis.

Several kinds of microbes are found in association with

[1] Monro and Workman, *Glasgow Medical Journal*, 1898, l., 343 *et seq.*

broncho-pneumonia. The most important are the *Pneumococcus*, the *Streptococcus pyogenes*, the *Staphylococcus aureus*, the *Staphylococcus albus*, and Friedländer's bacillus. The diphtheria bacillus may be found in cases secondary to diphtheria. Mixed infections are common.

Morbid Anatomy.—The disease begins in the finest bronchial tubes, so that at the outset it is a capillary bronchitis (p. 411). From the bronchioles the inflammation extends laterally into the lung tissue and onwards into the alveoli. Since each bronchiole, with its alveolar passage, infundibula and alveoli, is distributed to a lobule, the inflammation beginning in the bronchiole has a lobular arrangement. But, since many neighbouring lobules may be inflamed, a considerable mass of lung may be involved. The catarrh of the bronchiole is associated with the secretion of tough mucus and pus which obstruct the tube and often cause collapse of the lobule before the inflammation has reached it. As seen from the surface, collapsed portions are depressed and bluish-red. With or without a preceding stage of collapse, the inflammation extends from the bronchiole to the alveoli, and these become filled with catarrhal (epithelial) cells and leucocytes. The round-celled infiltration often extends to the peri-bronchial tissues.

The lungs are to a large extent crepitant, but are felt to contain nodules of consolidation. The bluish depressed areas of collapse are mostly seen towards the bases. The cut surface is dark-red. The inflamed patches are reddish-gray in an early stage, but afterwards become paler as in hepatisation. The small tubes contain tough secretion. Emphysema may be present in the upper lobes. In some exceptional cases, there is little or no distinct consolidation of tissue, and in others (*pseudo-lobar pneumonia*) the inflamed lobules may be so crowded together that a great part of a lobe is consolidated. The inflammation is apt to be specially intense in aspiration-pneumonia, and may proceed to softening and even gangrene.

Symptoms.—The clinical aspect varies much according to the cause of the attack. In the variety which follows measles or whooping-cough, convalescence is disturbed by

the renewal of fever, with cough and increased rapidity of the pulse. Breathing is difficult, as well as rapid and shallow. The alæ nasi move, the accessory muscles of respiration are active, and the lower part of the chest is drawn in with inspiration. The child is restless, anxious-looking, pale and livid, and may suffer from diarrhœa.

Physical Signs.—These, like the symptoms, are chiefly those of capillary bronchitis, viz., fine moist râles with some dry râles; but sometimes scattered patches of consolidation may be revealed by impaired percussion, bronchial breathing, and consonating râles.

Varieties.—In a large proportion of cases the onset and defervescence are gradual, and the course of the temperature is very irregular. When death occurs, it is usually in the second week, and is due to asphyxia resulting from the obstruction in the bronchioles.

In debilitated adult patients, the symptoms may not be well marked. There may be little more than pyrexia, cough, shortness of breath, and increase of the weakness. The condition may become associated with hypostatic pneumonia.

Primary cases have an abrupt onset with rapid elevation of temperature, and sometimes terminate by crisis. The physical signs as well as the symptoms may strongly suggest lobar pneumonia. Moreover, this type of the disease is due to the pneumococcus. These cases generally recover.

Diagnosis.—It may be difficult to distinguish between primary broncho-pneumonia of pseudo-lobar type and lobar pneumonia. The detection of a focus of disease in the less affected lung is in favour of broncho-pneumonia, but the course of the illness, or even an autopsy, will alone suffice to make the diagnosis certain. Another difficulty is to ascertain whether a broncho-pneumonia is tubercular, a possibility which must always be borne in mind. The sputum should be examined in doubtful cases for tubercle bacilli and elastic tissue. Continuance of signs of consolidation long after the acute symptoms have passed off, and still more, the development of signs of softening and excavation, are of grave significance.

Prognosis.—A considerable proportion of secondary cases in children are fatal. The mortality in the first five years of life is probably at least 20 per cent. Aspiration-pneumonia often goes on to suppuration or gangrene, and is very fatal. In primary cases the outlook is good. In rare instances broncho-pneumonia becomes chronic.

Treatment.—The patient should have a room at a temperature of 65° F. The atmosphere should be kept moist by a steam-kettle so as to make the sputum less tough. Ventilation is necessary, but there must be no draughts. Large warm poultices should be applied to different parts of the chest in rotation. Abundance of light food must be given, and the bowels should be gently moved each day. Ipecacuanha wine should be given as an expectorant, and if the secretions accumulate in the tubes, as an emetic also. If opiates are used, the greatest caution is required, owing to the risk of death by asphyxia. Strychnine hypodermically and ammonium carbonate internally may be used as stimulants. If the pulse threatens to fail, and the restlessness is great, whisky should be tried for its vaso-relaxant and narcotic effects. If the temperature is high, or if cerebral symptoms are very marked, the tepid bath or cold pack should be employed. A few cases may be saved by oxygen inhalations. In the tardy convalescence, great care must be taken to avoid fresh cold ; and cod-liver oil, chemical food, and a change of air may be highly beneficial.

vi. Chronic Interstitial Pneumonia

(Cirrhosis or Fibrosis of the Lung).

Strictly speaking, this is a rare disease, for while overgrowth of fibrous tissue is met with in many diseases of the lungs, these can generally be classified in a more satisfactory manner, as under the headings of tuberculosis, syphilis, pneumonokoniosis, etc. In the more limited sense, however, chronic interstitial pneumonia may arise in at least three ways. (1) Very rarely it follows acute lobar pneumonia. The alveolar walls become thickened, and the fibrinous exudation instead of undergoing softening and absorption

becomes organised. The naked-eye appearances suggest gray hepatisation, but the tissue is firmer and the cut surface smoother (*iron-gray induration*). If the disease progresses, the normal lung tissue undergoes atrophy, and bronchiectasis develops. (2) Broncho-pneumonia is a less rare cause, probably because the duration is longer, and because of the frequent occurrence of collapse and bronchial obstruction. Scattered patches of consolidation are produced in this way, and the condition may spread over a great part of the lung. (3) In a third group of cases, chronic inflammation spreads to the interstitial tissue of the lung from an inflamed pleura (*pleurogenic cirrhosis*). This fibrosis may be partial or general.

Alcoholism and syphilis have been regarded as predisposing to fibrosis, whatever the immediate cause may be.

Morbid Anatomy.—When the disease is advanced, the lung is greatly reduced in size, from contraction of the abnormally abundant fibrous tissue. It is firm, tough, smooth on section, and pigmented. The bronchi are dilated. The pleura is adherent and is usually much thickened. The disease is generally unilateral. If only a part of the lung is involved, it is usually the base which suffers. The opposite lung is enlarged. The right ventricle of the heart is hypertrophied.

Symptoms.—These may be a continuation of the symptoms referable to an inflammation of the lung or pleura, but frequently no such continuity can be traced. Shortness of breath and cough are the principal early symptoms. The sputum may be fœtid if bronchiectasis is present. There is sometimes hæmoptysis.

Physical Signs.—There is dulness on percussion, with increase of vocal fremitus and bronchial breathing, pointing to consolidation of the affected lung. The heart is displaced towards the affected side. The chest-wall is retracted and less mobile than normally, and the shoulder is drawn down on that side. There may be signs of bronchiectasis. The finger-ends may be clubbed.

Diagnosis.—The most important point is the distinction from fibroid phthisis, and this depends chiefly on the per-

sistent absence of tubercle bacilli from the sputum. The fact of both lungs being diseased is strongly in favour of tuberculosis.

Prognosis.—The patient may survive for many years, and for a long time enjoy fair health. Among the causes of death are general failure of nutrition, and failure of the right ventricle.

Treatment.—This includes good food and clothing; a favourable climate; precautions against a fresh attack of acute disease; counter-irritation over the diseased lung, and cod-liver oil internally. Bronchiectasis and various symptoms may demand appropriate treatment.

vii. Pneumonokoniosis.

The pneumonokonioses are diseases of the lungs due to inhalation of dust. Only the more important require to be mentioned here.

Anthracosis (*coal-miner's lung*) is not nearly so common now as formerly, the change being due to the excellent ventilation of mines now required by law. In former days colliers suffered much both from bronchitis and from phthisis. The lungs of colliers may contain large quantities of carbonaceous pigment (coal-dust, the soot of lamps and the products of combustion of explosives), and yet, since coal-dust causes but little irritation, there may be no evidence of actual disease. When anthracosis is present, the lungs are black and emphysematous, and contain small black nodules. When the lungs are cut, an inky liquid escapes. The nodules are masses of carbonaceous dust. The carbon, having entered the lymphatic system of the lungs through the walls of the alveoli, occupies that system and thus accounts for the black colour of the lungs. The carbon is usually arrested in the bronchial and other neighbouring glands, which are enlarged and black. It is to be noted first that coal-dust shows little tendency to cause interstitial overgrowth, and secondly that anthracosis (like any other pneumonokoniosis) predisposes a lung to tubercular infection.

Symptoms.—These point to, and are largely the result of, chronic bronchitis and emphysema. There is cough, with

black expectoration, and shortness of breath on exertion. After a time, if the lungs become tubercular, emaciation and other symptoms of ordinary phthisis may be added to those of bronchitis.

Lithosis (*silicosis* or *chalicosis ;* including *stonemason's phthisis*, *grinder's rot* and *potter's rot*) results from inhalation of siliceous dust. The dust is very irritating, and causes well-marked fibrosis with the development of hard nodules which contain gritty material. The character and duration of the symptoms vary much in different industries ; phthisis in some, and bronchitis or asthma in others, being the prevailing type.

Siderosis is the disease which results in colour-grinders from the inhalation of oxide of iron. The lungs are red and fibrotic. The symptoms are those of rapid phthisis.

Prognosis.—Most of the pneumonokonioses progress so slowly that the sufferer can continue his work for years. In all of them there is the risk of tubercular infection. Sometimes, however, pneumonia or hæmoptysis causes death early.

Treatment.—If the disease is in the early stage, the patient should give up his occupation ; otherwise the treatment is similar to that of phthisis or chronic bronchitis.

viii. Pulmonary Emphysema.

Pulmonary emphysema may be either interlobular or vesicular.

In Interlobular or Interstitial Emphysema air is present in the connective tissue of the lung. This may result from a wound of the lung—*e.g.*, by a broken rib—or it may be due to acute over-distension of the air vesicles by violent expiratory efforts, as in whooping-cough or diphtheria. In either case, vesicles are ruptured, and air escapes into the surrounding tissues. The air may pass from the root of the lung to the subcutaneous tissue of the neck. It can be seen under the pleura in the form of beads or bullæ.

In Vesicular Emphysema, the air vesicles are over-distended, and secondary changes are brought about.

Etiology.—Three varieties must be distinguished :

(1) *Expiratory* (*substantive*, *substantial*, *idiopathic*, *hypertrophic*, *large-lunged*). In expiration with the glottis closed, the lungs are compressed by the floor and walls of the chest, and the air within the lungs exerts a distensile force on all the air-sacs. At certain parts—*e.g.*, the anterior margins, and above the clavicles—the lungs are not so well supported by the chest-wall as elsewhere, and at these parts over-distension of the air-sacs is most readily produced. Violent expiration with closed glottis is the principal element in coughing, so that this variety of emphysema is met with in chronic bronchitis and in other diseases accompanied by severe coughing over a long period. In addition to the increased air-pressure, importance must be attached to diminished resisting power of the lung tissue, and the latter may be attributed to chronic catarrh, senility, and inherited predisposition. The emphysema of glass-blowers and of players upon wind instruments is doubtless mainly expiratory.

(2) *Inspiratory* (*compensatory*, *complementary*, *vicarious*). If one part of a lung does not expand fully in inspiration, some other part will be over-distended, or the chest-wall will be drawn in, or both of these things may happen. Thus the imperfect expansion, or local diminution of bulk, seen in the collapse of broncho-pneumonia, in obsolete tuberculosis, and in fibroid phthisis is associated with emphysema. The emphysema may be localised in the neighbourhood of the lesion of which it is a consequence. General adhesion of the pleura frequently leads to marked emphysema of the anterior margin of the lung.

(3) *Senile* (*atrophic*, *degenerative*, *small-lunged*). This is a primary atrophy of the lung tissue which accompanies senile atrophy of the other tissues of the body. The lungs in this form are reduced in bulk. The changes in the air vesicles are similar to those in the first variety. The senile and substantive forms will naturally often be present in the same individual.

Morbid Anatomy.—From the practical or clinical point of view, it is chiefly the first variety of emphysema, the *sub-*

stantive or *hypertrophic form*, which requires consideration. The chest is barrel-shaped. The lungs are much enlarged, and have a soft ~~downy~~ doughy feel. The emphysematous parts take the form of air-filled bullæ which are sometimes of great size and are specially noticeable at the apex, anterior margin and base. Microscopic investigation shows that the infundibula and alveoli become more and more distended by the air-pressure within them, until the intervening septa undergo atrophy, become perforated, and are finally destroyed, and neighbouring cavities coalesce. By a continuation of the process, large air-filled bullæ are at length produced. The destruction of the bloodvessels in the partitions, when the disease becomes extensive, causes serious obstruction to the pulmonary circulation, so that the right ventricle becomes hypertrophied. The atrophy of the elastic tissue prevents the lung from collapsing as in health. The destruction of alveoli diminishes the respiratory capacity. As the morbid change advances, the right heart becomes dilated, and general venous congestion supervenes.

Symptoms.—Chronic bronchitis is usually present, with cough and spit. In addition, the emphysema gives rise to shortness of breath. At first this is only on exertion ; later on it becomes more continuous, and in advanced cases amounts to orthopnœa. Lividity is another feature, and is often associated with pallor. There may be asthmatic attacks. Owing to the lessened elasticity of the lungs, expiration is especially embarrassed.

Physical Signs.—The chest is barrel-shaped, and increased in capacity—a permanent and exaggerated condition of full inspiration. The gait is stooping. The apex-beat is feeble or inappreciable, the area of superficial cardiac dulness greatly diminished or altogether lost, and the sounds of the heart faint. A tricuspid systolic murmur may be audible. Percussion over the lungs is highly resonant (hyper-resonant), and the upper border of hepatic dulness may be much depressed. The respiratory murmur is often faint, sometimes harsh ; and its expiratory portion is prolonged. The dry and moist râles of bronchitis may be present. If the right ventricle has failed, enlargement of the liver, dropsy,

and other phenomena of venous engorgement will be present.

Prognosis.—It is the presence of chronic bronchitis which makes ordinary emphysema a progressive disease. Each recurrence of winter-cough leads to further permanent damage to the lung tissue, diminishing the respiratory surface and increasing the strain upon the right ventricle. With temporary interruptions, the patient may do much work during many years, but in the long-run the tendency is to death by right ventricle failure, which may be precipitated at any time by an exacerbation of the bronchitis.

Treatment.—This is similar to that which is adopted for chronic bronchitis. Every precaution must be taken to avoid fresh colds. When the right ventricle becomes disabled, as shown by dropsy, lividity and orthopnœa, appropriate measures must be employed, including a mercurial purge, diaphoretics, oxygen inhalations, and in some cases blood-letting, followed by cardiac tonics and stimulants.

ix. Pulmonary Gangrene.

Etiology.—In this condition there is necrosis of lung tissue followed by putrefaction. If foreign matter gets into the bronchial tubes—*e.g.*, a piece of bread, or vomited matter, or a tooth accidentally dislodged in course of an operation under chloroform, or septic discharges from lesions of the mouth, or from ulcers or abscesses which perforate into the air-passages—the result is likely to be an acute bronchitis with putrefaction of the secretion ; and the putrid secretion often causes aspiration-pneumonia which goes on to gangrene. The contents of a tubercular or bronchiectatic cavity may decompose and cause gangrene. An aneurysm or other tumour pressing upon the root of the lung may cause bronchiectasis and gangrene. Gangrene also occurs in connection with metastatic abscesses, and occasionally follows the hæmorrhagic infarction, the pneumonia of drunkards, diabetics and other debilitated subjects, or a severe attack of a specific fever. Sometimes no definite cause can be ascertained. Micro-organisms are of course the immediate

agents in producing the changes, especially the *Staphylococcus pyogenes aureus* and *albus*.

Morbid Anatomy.—Two forms are recognised. In *diffuse* gangrene, patches are present throughout a great part of one lung, or throughout the whole of one or both lungs ; while in the *circumscribed* form, the gangrene is very localised. The decomposing slough has a greenish colour, and as it softens it gives rise to a cavity with ragged walls and fœtid contents. Immediately around the gangrene there is a zone of intense inflammation. The dead tissue may become detached by the inflammatory process, so that a suppurating cavity is left, and if this is small, it may contract and give place to a cicatrix. Occasionally the slough involves the pleura, in which case pneumothorax and pyopneumothorax may result. Another occasional result is hæmorrhage. The decomposing juices from the slough induce an intense bronchitis, and the secretions which are poured out may cause an aspiration-pneumonia in various parts of the lungs, and thus lead to diffuse gangrene.

Symptoms.—The patient is usually under observation with pulmonary symptoms, but gangrene can scarcely be diagnosed until the putrid juices from the slough reach a bronchus and are evacuated in the sputum. The horrible fœtor of the spit and breath is the most characteristic evidence of gangrene. The sputum contains lung tissue, triple phosphates, margarine crystals, pus and bacteria. On standing, it tends to separate into three layers. The lowest is dense and purulent, has a foul smell, and contains fatty acids, bacteria and elastic tissue, but not Dittrich's plugs. The middle layer is serous and mucous, and comparatively clear. The upper layer is frothy. Blood is frequently present, and indeed the involvement of a considerable vessel may lead to fatal hæmorrhage. There is often severe prostration, with fever and a rapid pulse.

Physical Signs.—These may not be distinctive, or they may be masked by those of the primary disease. So far as they are characteristic, they point to softening and excavation in the midst of an area of consolidation.

Prognosis.—Death usually results from exhaustion, but recovery may take place. The diffuse variety is naturally much more serious than the circumscribed.

Treatment.—Good feeding and nursing are important. Stimulants will often be required. Antiseptic inhalations should be employed. If the gangrenous focus can be localised, the surgeon may occasionally render useful aid by draining the cavity.

x. Pulmonary Abscess.

Etiology.—In rare cases of lobar pneumonia and of tuberculosis, the diseased portion of lung becomes the seat of one or of numerous abscesses. Aspiration-pneumonia often goes on to abscess formation (which in many cases cannot be definitely separated from gangrene). In pyæmia, there are numerous small metastatic abscesses, mostly situated at the surface of the lung, of a grayish colour and surrounded by intensely congested tissue. Abscess may also result from suppuration of a hydatid cyst, or from rupture into the lung of an abscess in some other part, as for instance hepatic abscess, mediastinal abscess, a suppurating bronchial gland, or empyema.

Symptoms.—These vary greatly in different cases. If the abscess does not communicate with a bronchus, the constitutional symptoms of suppuration may be present, with few or no abnormal physical signs. If the suppuration follows pneumonia, the signs of softening may be recognisable. In pyæmia, the general symptoms are most important, but there may be physical signs of broncho-pneumonia. When the abscess communicates with a bronchus, there are cough and expectoration. The latter may resemble pure pus or may be chocolate-coloured. It may have little or no odour, or may smell most offensively. It should be carefully examined for lung tissue, liver tissue, bile, or other evidence of the original seat of abscess formation.

Physical Signs.—Apart from those which may be due to the primary disease, there may be physical signs pointing to a localised consolidation. If the abscess bursts and discharges, the signs of cavity will speedily develop. In

pyæmia, the physical signs are likely to be those of broncho-pneumonia, unless pleurisy be also present.

Prognosis.—Recovery occasionally takes place, either spontaneously or after operation.

Treatment.—If the abscess can be localised, it ought to be incised and drained. Otherwise, feeding, nursing, stimulants and tonics such as quinine and iron are the indications.

xi. Tumours of the Lung.

Primary tumours of the lung are not nearly so common as secondary growths. *Primary* tumours are generally confined to one lung. The most common is cancer originating in the epithelial lining of the respiratory tract and extending to the bronchial glands and often to the pleura. Of *secondary* growths, one of the most common is a lymphosarcoma originating in the bronchial glands and invading the lung by way of its root. The lung thus comes to be largely replaced by tumour tissue which has a white or gray colour, and may be softened in various places. By compressing the bronchi, it may lead to bronchiectasis. Sarcomas and cancers originating in the alimentary tract and in various other parts of the body may give rise to embolic growths in the lungs. These are generally multiple and distributed in both lungs. When the pleura is involved in malignant disease of the lung, the accompanying exudation is sometimes hæmorrhagic. The bronchial glands are usually, and the cervical glands frequently, involved by the disease. These tumours are most common about middle life.

Symptoms.—These are not always characteristic. There may be pain and tenderness in the chest. Cough is present, and may be stridulous if the air-passages are compressed, or brassy if the laryngeal nerves are implicated. Expectoration is usually scanty, and sometimes resembles red-currant jelly. Œdema of the upper part of the trunk and upper limbs may result from pressure upon veins, and the surface veins may be enlarged. The swelling may be accompanied by lividity. Dyspnœa may result from destruction of lung tissue, pleural effusion, pressure on the air-passages, or pressure on nerves. There may be pyrexia at times.

Physical Signs.—There may be local bulging of the chest-wall, or the latter may be perforated by the growth. Percussion is dull over a large growth. If the latter is in contact with a bronchus, bronchial breathing may be heard over the growth. If the tumour compresses the bronchus, the respiratory murmur may be enfeebled or lost on the corresponding side of the chest. Enlarged glands are sometimes found in the neck.

Diagnosis.—This is easy if a primary growth has been previously recognised elsewhere. The insidious onset of the symptoms and signs above described, and the existence of red-currant jelly expectoration and of hæmorrhagic pleural effusion are important evidence in favour of tumour. Marked dulness on percussion without displacement of the heart is suggestive of tumour rather than of pleural effusion, but it is advisable to explore with a hypodermic needle. Even if pleural effusion is present, the fact is not sufficient to exclude tumour. Constant recurrence of the effusion after removal by aspiration is ominous, especially if the liquid be blood-stained.

Prognosis.—The disease tends gradually to cause fatal exhaustion, but the immediate cause of death may be involvement of the heart or of some other important structure. The average duration of life may be put at about a year from the time when the disease is recognised.

Treatment is purely palliative. It may be necessary to aspirate the pleural cavity for the relief of urgent dyspnœa.

DISEASES OF THE PLEURA.

i. Pleurisy (Pleuritis).

The pleura suffers from inflammation more frequently than the other serous membranes, and the inflammation is met with in many forms of which no satisfactory classification is available. Thus pleurisy may be acute, subacute or chronic ; primary or secondary ; general or localised ; dry or accompanied by effusion, and the effusion may be sero-fibrinous, purulent or hæmorrhagic.

General Etiology.—Pleurisy occurs at all ages, but is most

common in middle life. The cause may be local or general. The *local* causes include injury or disease of the chest-wall, such as fractures of ribs or caries of the spine, and the entrance of irritating matter into the pleural cavity from the lung or elsewhere, as in phthisis, pneumonia, etc. The *general* causes are also varied. Among the most common are the pneumococcus, the tubercle bacillus, the streptococcus, and the rheumatic infection. A chill is often the apparent cause, but the tendency now is to regard this as merely the predisposing factor, and to suppose that some microbe, and especially the bacillus of tubercle, is the exciting cause (see p. 444, under Sero-fibrinous Pleurisy). Pleurisy occurring in association with scarlet fever, influenza, enteric fever, Bright's disease, septicæmia and pyæmia is attributable to the specific microbes of the disease or their toxins, or to some morbid blood state, or to a secondary infection—*e.g.*, by the pneumococcus. Pleurisy is sometimes secondary to peritonitis, the irritant travelling through the diaphragm by the lymph channels which pass from the one sac to the other. Multiple serous membrane inflammation is known as *polyorrhomenitis*.

The principal organisms found in inflammatory pleural effusions are the same whether the effusions are purulent or non-purulent. In the latter case, however, the microbes are so inconstant or so scanty that the exudations are often found to be sterile. The microbes include the pneumococcus, the tubercle bacillus, the streptococcus, and the staphylococcus. Sometimes the infection is mixed.

Pleurisy with Fibrinous Exudation (Dry or Plastic or Adhesive Pleurisy).

In this form of pleurisy the exudation is so scanty as not to give rise to the physical signs of fluid effusion.

Etiology.—Dry pleurisy may follow a chill, it is generally present in phthisis, it is common in rheumatism and Bright's disease, and it is present when pneumonia and other morbid processes involve the surface of the lung. It may be caused by the pneumococcus, though there is no pneumonia. The pericardium and both pleuræ may be simultaneously in-

flamed (polyorrhomenitis) through the agency of the pneumococcus.

Morbid Anatomy.—The inflammation may involve a small or large portion, or the whole of the pleura. Where the cause is general (rheumatism, chill, pneumococcus infection of the pleura), the lower part of the pleura is apt to suffer chiefly or solely, whereas pleurisy due to local disease of the lung or thoracic wall may be localised anywhere. Thus localised pleural friction at the apex is often one of the earliest physical signs of phthisis.

There is first hyperæmia, and this is followed by the fibrinous exudation, of which there may be either a thin or a thick layer. The usual termination is by adhesion of the inflamed surfaces and permanent fibrous-tissue union. In the tubercular variety there is specially apt to be considerable thickening of the pleura.

Symptoms.—There may be no definite symptoms; or there may be pain, slight pyrexia and cough without expectoration. Severe pain, in the form of a stitch, may be induced by breathing deeply, or by coughing. The pain may correspond to the seat of inflammation, or may be referred to the distribution of irritated intercostal nerves—*e.g.*, to the front of the abdomen.

Physical Signs.—Auscultation may reveal friction sound, which, as just explained, is sometimes at a distance from the seat of pain. As the patient breathes quietly to avoid a stitch, the respiratory murmur may be feeble. If the pleura undergoes thickening, the percussion note and respiratory murmur will be impaired.

Diagnosis.—*Pleurodynia* or rheumatism of the intercostal muscles is distinguished by the absence of shivering, of fever, and of friction sound. In *intercostal neuralgia*, there is no fever or friction sound, movement has no great influence on the pain, and the patient is probably predisposed to neuralgia by anæmia, neurotic tendency, or otherwise.

Prognosis.—Considered apart from its cause, dry pleurisy, especially if localised, is not a serious matter.

Treatment. — Little treatment is necessary. Morphine may be given hypodermically at the outset, or leeches may

be applied, if the pain is severe. For continued pain or cough a blister applied locally is a suitable remedy. Later on tonics and a change of air are indicated. Any disease to which the pleurisy is secondary must receive due attention.

Pleurisy with Sero-fibrinous Exudation.

No definite line of demarcation can be drawn between this and the preceding variety from the anatomical point of view, but clinically the sero-fibrinous form is characterised by physical signs which point to the presence of liquid in the pleural cavity. Puncture of the chest by an aspirator or hypodermic needle enables the nature of the fluid to be recognised with certainty, although this can generally be suspected from the history and symptoms.

Etiology.—This disease often follows a chill, but there is a general tendency now to regard this as being merely the predisposing cause, and to look upon the tubercle bacillus as the actual excitant of the inflammation in the majority of primary acute pleurisies such as were recently supposed to be 'simple' or 'idiopathic.' The pneumococcus and streptococcus are also important causes.

With regard to the tubercular nature of the so-called simple acute pleurisies, these must of course be distinguished from the secondary non-tubercular pleurisies met with in phthisis, where not the bacilli but the toxins pass through the pleural membrane to reach the cavity of the sac. Again, in general tuberculosis, miliary tubercles may be found on both visceral and parietal layers of the pleura. But the simple acute pleurisies particularly referred to here are a third group in which the bacillus attacks the pleura first of all the organs or tissues of the body—at least, in such a way as to give rise to marked symptoms. Some of the evidence for the tubercular nature of the disease may be stated thus: (1) In some cases there has been anatomical evidence, tubercles having been found after death in acute cases which during life were regarded as simple. (2) It is sometimes possible to detect tubercle bacilli during life by withdrawing some of the exudate, allowing this to clot, and

then digesting and centrifugalising. (3) While inoculation of small quantities of serum from the pleural exudation frequently fails to infect animals with tubercle, the use of larger quantities (ʒss. or more) is often followed by infection. The bacilli are probably scanty, or deposited at the bottom of the exudation, or buried in the lymph ; and for the same reason, ordinary cultures made from the exudation show no growth of tubercle bacilli. (4) A considerable proportion of these cases ultimately succumb to tuberculosis of the lungs or other organs. At the same time, the assertion[1] that ' however rapidly and apparently completely the victims of such pleurisies recover, statistics prove that the majority of them die before three years are over with undoubted tuberculous disease of the lungs and other organs,' is, I consider, exaggerated. Either the proportion must be reduced or the period of observation must be lengthened. Osler quotes the recent figures of Hedges, who found that, of 130 cases followed for seven years, 40 per cent. became tubercular. (5) The tuberculin test, the agglutinin test, and cultivation on peptone jelly containing blood have furnished evidence in the same direction. Nevertheless, if the conditions of life are favourable, scarcely any form of tuberculosis is so curable as that which is limited to the pleura.

Morbid Anatomy.—The disease is generally unilateral. Fibrin is present as in dry pleurisy, and in addition there is liquid effusion varying in amount up to several pints. This serum is greenish-yellow, alkaline and highly albuminous. The cells are mostly mononuclear. In many cases the pneumococcus will be found, but in the tubercular cases, the usual tests almost always show that the fluid is sterile. If the effusion is large, the lung is collapsed, and neighbouring organs are displaced.

Symptoms.—The disease often sets in suddenly, with or without a preceding chill, in a person who has been previously in perfect health. The early symptoms are a stitch in the side, dry cough and repeated shivering. The pain is often of a severe stabbing character, and is aggravated by

[1] J. A. Coutts in Allchin's ' Manual of Medicine,' i. 225.

deep breathing, coughing, and movement in bed, as well as by pressure over the seat of lesion. Often, however, the onset is insidious. There is moderate fever, which usually subsides in a week or so by lysis, though occasionally the pyrexia continues for many weeks. The patient at first tends to avoid lying on the painful side, but after considerable effusion takes place and prevents the inflamed pleural surfaces from rubbing against one another, he rather prefers this side, since freer movement is thereby permitted to the healthy side of the chest. Respiration is accelerated and shallow in the early stage, on account of the pain, and in some cases later on, on account of great effusion. As a rule, however, even with abundant effusion, there is no dyspnœa as long as the patient is resting.

Physical Signs.—Before the liquid effusion has been poured into the thorax, friction sound is heard over the inflamed pleura. When effusion is present, friction may still be heard above the level of liquid effusion. If the effusion is scanty, the principal signs are dulness on percussion, enfeeblement of respiratory murmur, and diminution of vocal fremitus and vocal resonance at the posterior base. Two fingers' breadth of dulness at the base is said to correspond to 15 ounces of effusion.

It should be remembered that the intrathoracic pressure is normally negative to such a degree that a large amount of fluid can be poured into the pleural sac before positive pressure is exerted on neighbouring structures. Before there is enough of fluid to neutralise the negative pressure, the lung on the affected side is permitted to collapse by its own elasticity, and the pericardium and heart are allowed to be drawn over (unless previously fixed by adhesions) by the elasticity of the structures on the opposite side of the chest. At or just before the time when the fluid becomes sufficient to neutralise the negative pressure, the following *four cardinal signs* can be recognised, viz., dulness on percussion and loss of vocal fremitus over the effusion, displacement of the heart towards the unaffected side, and alteration in the respiratory murmur. The dulness does not change much with the posture of the patient. When the patient sits up, the upper limit of dulness

is not a horizontal line, but a parabola. It extends from, say, the third costal cartilage upwards and outwards, and reaches its highest level in the axilla. It then sinks downwards and inwards at the back. Above the level of dulness in front there is tympanitic percussion (Skodaic resonance). The breath-sound and voice-sounds are usually lost over the effusion, but sometimes there is tubular breathing, with bronchophony or pectoriloquy, a circumstance which makes the cardiac displacement and loss of vocal fremitus exceedingly important in diagnosis.

Less important signs are : increased circumference of the chest, alteration in the shape of the chest, ægophony (a bleating quality of the voice-sounds as heard through a layer of pleural effusion), and undue fulness of the intercostal spaces. According to Baccelli, the whispered voice is transmitted through a serous but not through a purulent effusion (*Baccelli's sign*).

It is to be noted that even with a large pleural effusion on the left side, the stomach note may be obtained as high as the sixth rib in the nipple line, and that with a similar effusion on the right side, the liver may not be appreciably depressed. At the same time, since the pleura extends further down than the lung, there is a tendency for the dulness due to a large left-sided effusion to encroach upon Traube's semilunar space, viz., that portion of the area of gastric percussion-note which is bounded by the lung and liver above, and the lower costal margin below.

Pressure Effects.—If the fluid accumulate to a further degree than has been indicated thus far, it will begin to exert positive pressure. The dulness rises above the third rib, sometimes to the extreme apex, and Skodaic resonance disappears. The diaphragm and abdominal organs are displaced downwards, a systolic murmur may develop over the base of the heart in consequence of straightening or increased bending of the great vessels, and the strain upon the active lung may be manifested by moist râles, hæmoptysis, dyspnœa and cyanosis.

Diagnosis.—This can usually be made with ease if sufficient care be taken. *Pneumonia* is the most important

source of confusion, and is as a matter of fact accompanied at times by pleurisy with effusion. In pneumonia, the onset with a rigor, the rapid respiration, the rusty sputum, the absence of chlorides from the urine, the course of the temperature, and the characteristic physical signs are important; whilst in pleural effusion there are the cardiac displacement and loss of vocal fremitus. A well-marked leucocytosis points to pneumonia or empyema as against sero-fibrinous pleurisy. In any case of doubt, a sterilised hypodermic needle should be inserted into the chest. The exploratory syringe further indicates the character of the liquid effusion, whether sero-fibrinous, purulent, or hæmorrhagic.

Bacteriological examination may give the key to the microbic origin. The pneumococcus or some other organism may be found in cultures; but if cultures are sterile, the case is to be regarded as probably tubercular. If organisms are not found in the exudate, Jousset's method of *inoscopy*[1] ought to be employed. Some ounces at least of the effusion are withdrawn from the pleural cavity and allowed to clot. The clot which entangles the microbes is then digested in a pepsin and hydrochloric acid mixture,[2] which dissolves practically everything except the solid bacilli, and perhaps for a time the cells. The bacilli are separated by thorough centrifugalisation, and are then demonstrated by staining in the usual way.

The method of *cytodiagnosis* has been largely employed in recent years, but it cannot yet be regarded as an absolutely reliable guide. It is generally recognised that a preponderance of lymphocytes favours a diagnosis of tuberculosis; but it has been suggested, on the one hand, that polynuclear cells indicate an early stage, and mononuclear cells a late stage of the inflammation, whatever be the organism that caused it; and it has been asserted, on the other hand, that in the case of tubercular effusions at least,

[1] *ἴς, ἰνός*, fibre.

[2] Pepsin, 2 grammes; sodium fluoride, 3 grammes; hydrochloric acid, 10 c.c.; glycerin, 10 c.c.; distilled water, 1,000 c.c. (Boston, 'Clinical Diagnosis,' p. 492).

while the mononuclear cells are the most characteristic, these may in late stages come to be in the minority. In septic pleurisies the cells are mostly polymorphonuclear, but a preponderance of polymorphous cells does not necessarily indicate that the pleurisy is septic. In the non-inflammatory effusions of renal and cardiac disease, the cells are likely to be very few in number, the characteristic ones being flat endothelium from the pleura itself, and occasionally united together in plates.

Prognosis.—So long as there is no positive pressure in the chest, the immediate prognosis is good. The effusion may, after a few weeks, tend to undergo absorption ; sometimes quickly, but often very slowly. It may reaccumulate, in spite of repeated tappings. The ultimate outlook is always attended by some anxiety on account of the relationships existing between pleurisy and phthisis. There is no doubt that many of these cases ultimately die of phthisis. Sometimes pleurisy represents the first invasion of the body by the bacillus ; in other cases, the microbe has previously attacked the lung ; and in yet other cases, it may be, the collapse of the lung by the effusion, the febrile illness, and the confinement to an ill-ventilated room have predisposed the lung to tubercular infection.

In rare cases of pleurisy with effusion, death occurs suddenly from syncope. The effusion is not always excessive in such cases, and the cause of death is not yet clearly understood, though various explanations, such as kinking of the inferior vena cava, pulmonary embolism or thrombosis, degeneration of the heart, and œdema of the opposite lung, have been suggested.

Treatment.—Rest in bed, fever diet, a diaphoretic mixture and a laxative are indicated at the outset. If the pain is severe, morphine should be given hypodermically, and poultices, leeches, or an ice-bag may be applied locally. Some authorities recommend withdrawal of the effusion as early as possible, the operation being repeated as soon as the fluid reaccumulates ; but this causes the patient a good deal of suffering, and its wisdom is doubtful so long as there is no positive pressure in the chest. If such pressure does

exist, the chest must be tapped at once to remove a danger to life. If it does not exist, it is good practice to wait for two, or at most three, weeks, and then to aspirate for the sake of the collapsed lung, unless the fluid is rapidly diminishing of its own account. If, however, tuberculosis of the lung already exists on the affected side, it is better to postpone aspiration even longer, and sometimes to avoid it altogether, since experience shows that the presence of effusion will sometimes retard or arrest the tubercular process.

If there is doubt at any stage as to the character of the fluid, a hypodermic syringe should be used to withdraw a small quantity, and if it proves to be pus, arrangements must promptly be made to thoroughly drain the cavity. If, however, the effusion is serous, the physician may wait for a day or two to see if absorption does not speedily follow the removal of the syringeful of fluid, an occurrence which is quite familiar ; or the aspirator may be at once employed to withdraw slowly as much fluid as can be removed with comfort to the patient. The onset of faintness, of coughing, of spitting of blood, or of pain due to irritation of the visceral pleura by the point of the aspirator-needle, is an indication that the operation should be desisted from. If the fluid ceases to flow, the needle must be withdrawn. If there is reason to believe that this is owing to the needle being blocked by lymph, it may, after being cleaned, be inserted at another spot. The needle must not be stirred about inside the chest in the hope of hitting upon more fluid effusion.

Different physicians prefer different sites for the puncture. The eighth space in the line of the scapular angle is very convenient. Or the sixth or seventh space in the mid-axillary line, with the patient lying down, may be selected (in children, on the right side, the fifth space, on account of the high position of the liver). The lowest level of the effusion is to be avoided, as flakes of lymph settle there and obstruct the needle. The skin should be disinfected and frozen with ice and salt, or with ethyl chloride, just before the operation ; and the puncture should be sealed with collodion after the needle is withdrawn. In rare instances

albuminous expectoration follows the tapping. It is associated with dyspnœa, and may prove rapidly fatal.

Barr recommends that at the time of the tapping adrenalin chloride (1 drachm of 1 in 1,000 solution) should be injected into the chest to prevent reaccumulation, and that sterilised air should also be introduced to diminish the tendency to the formation of adhesions.

After two or three days, the patient should be allowed to go about freely. Stimulating liniments may be applied over the chest where any dulness still remains. Pulmonary gymnastics should be begun soon; the patient may blow water from one Wolff's bottle to another, or he may practise on a wind instrument, if this can be done without causing offence to others. Naunyn causes his patient to sit on a chair, and grasp it firmly with the hand of the sound side; by pressing the corresponding arm firmly against the chest and making strong inspiratory efforts, the affected side is forcibly expanded. Rosenthal disapproves of exercises of this kind; he makes the patient practise calm breathing by the nose, beating time with his hand. Later on the patient lies on his back and breathes twenty times, beating time as before; and then he gives twenty diaphragmatic respirations, lifting the physician's hand laid on the abdomen. Still later the respirations are made as the patient lies on his back, with one hand grasping the head of the bed; with the arm held out from the body; with both hands holding the head of the bed; and, finally, with passive extension of the flexed elbows during inspiration.

A change of air, cod-liver oil and tonics are desirable, and every precaution must be taken for some years at least to maintain the best possible vigour so as to resist any fresh attack of the tubercular virus. In chronic cases, where repeated tappings fail, free incision and drainage are justifiable.

Pleurisy with Purulent Exudation (Empyema).

Etiology.—An ordinary sero-fibrinous pleurisy sometimes becomes purulent in children, rarely in adults. Empyema also follows infectious diseases, such as scarlet fever, enteric,

pneumonia, pyæmia, and puerperal septicæmia. It may also arise from local causes, such as perforation of the pleura in phthisis. The principal microbes are the pneumococcus, streptococcus, and tubercle bacillus.

Morbid Anatomy.—The pus has various degrees of turbidity, and is sometimes sweet and sometimes foul-smelling. After a time, there is great thickening of the pleural layers, and the lung may be collapsed to very small bulk. Sometimes the pus is confined by pleural adhesions to a part only of the pleural cavity (*loculated* or *encysted* empyema).

Symptoms.—These do not differ greatly from those of sero-fibrinous effusion, and are often insufficient by themselves for diagnosis. The onset is often insidious, and chest symptoms may be almost absent. The patient may have had what appeared to be an ordinary attack of pneumonia, but after the defervescence he fails to regain his strength, and after a time the thorax is found to be filling with fluid. Hectic fever, sweating, rapid emaciation, and a distinct leucocytosis are very suggestive of a purulent effusion. Œdema of the chest-wall is sometimes present, and may be regarded as pathognomonic of purulent as distinguished from sero-fibrinous effusion.

Physical Signs.—These are similar to those of sero-fibrinous effusion. Bulging of intercostal spaces, however, is apt to be greater in empyema, probably because the toxins have a more paralysing effect on the intercostal muscles. *Pulsating pleurisy* (see below) is almost always purulent. *Baccelli's sign*, the alleged feebleness of the voice-sounds over a purulent as compared with a sero-fibrinous effusion, is not reliable.

Diagnosis.—This depends on the history of the case, the special symptoms just mentioned, the signs of pleural effusion, and the result of exploratory puncture.

Prognosis.—The immediate outlook is much more serious than in the case of non-purulent effusion. Most cases, if left to themselves, end in death; this may be due to syncope, asthenia, pyæmia, rupture into the pericardium or peritoneum, etc. Quite exceptionally spontaneous recovery takes place. Either the pus may dry in and become infil-

trated by lime salts, or it may escape through the lung, or into a bronchus, or through the chest-wall. An empyema which perforates the chest-wall is called *empyema necessitatis*.

Treatment.—This consists in the prompt and complete evacuation of the pus by surgical methods, though it occasionally happens that recovery takes place in childhood after a single tapping. Respiratory gymnastics should be begun early, to promote the expansion of the lung and the obliteration of the cavity.

Pleurisy with Hæmorrhagic Exudation.

This variety of pleurisy may be due to the tubercle bacillus or to some other organism. It occurs, too, in connection with cancer of the pleura. The pleurisy of cachectic states and of hepatic cirrhosis may also be associated with hæmorrhage; but, as Osler points out, such pleurisies are frequently tubercular. The fluid removed by the needle is more watery than pure blood.

Special Varieties of Pleurisy.

Diaphragmatic Pleurisy.—Pleurisy strictly limited to the diaphragmatic surface of the lung is not common. The suffering may be intense, and yet there may be no abnormal physical signs. The patient sits erect, breathes rapidly, and complains of pain at the lower ribs of one side, sometimes darting to the back or shoulder. The pain may suggest acute abdominal disease. There may be tenderness over the seat of lesion. Diaphragmatic pleurisies may be secondary to disease in the abdomen, involving the peritoneum, liver or spleen.

Fibroid Pleurisy.—With or without a preceding attack of acute inflammation, the pleura occasionally undergoes a gradual increase in thickness, and by its contraction damages the lung. The fibrosis involves at the same time the connective tissue of the lung itself. Such cases are often, if not always, tubercular.

Encysted Pleurisy.—If inflammation attacks a pleura whose cavity is partly obliterated by old adhesions, the fresh exudation will be localised or encysted in one or

perhaps several situations. Pleurisies of this kind which come under notice (*e.g.*, *diaphragmatic*, *interlobar*, or *axillary*) are generally purulent. Repeated punctures may be required both for diagnosis and for treatment.

Pulsating Pleurisy.—In this variety the pulsations of the heart are communicated by the fluid to the chest-wall. Pulsation of a pleural effusion is rare, and is almost, but not absolutely, confined to the left side of the chest and to purulent effusions (*pulsating empyema*). An *empyema necessitatis* may pulsate, and may thus closely resemble an aneurysm.

ii. Hydrothorax.

Hydrothorax or (non-inflammatory) dropsy of the pleural cavity is often part of a general dropsy, whether cardiac, or renal, or due to a morbid blood state. It may also result from a local cause, such as pressure upon veins by a tumour. Except in the last case, it is usually bilateral, but it is seldom present to the same extent on both sides. In cardiac dropsy it is chiefly the right thorax that suffers, possibly in consequence of pressure on the azygos veins. The diagnosis is rarely difficult. There is dulness on percussion, with impairment of breath-sounds, of voice-sounds and of vocal fremitus. But there is no friction sound, and, moreover, dropsy is likely to be present elsewhere.

The condition is not, as a rule, serious of itself, but if the effusion is at all considerable, its removal may give great relief to the patient, and do much to promote recovery. If diuretics and purges fail, aspiration should be resorted to, and this operation may, if required, be repeated for an indefinite number of times. The primary condition will require attention much more than the hydrothorax.

iii. Hæmothorax.

This name is not properly applicable to inflammatory effusions which contain blood, such as have been described under the head of pleurisy. Actual hæmorrhage into the pleural cavity is generally due to injury of the wall or contents of the chest. It also results from rupture of an

aneurysm, an occurrence which is likely to cause speedy death. Rapid hæmorrhage will cause corresponding general symptoms, while a gradual bleeding will give rise to the usual signs of pleural effusion. Unless threatening symptoms are present, the effusion may be left alone. If puncture is required, great care must be taken to insure asepsis.

iv. Pneumothorax, Hydropneumothorax, and Pyopneumothorax.

Etiology.—Air may obtain access to the thorax through a lesion either of the chest-wall or of the lung. The great majority of cases are connected with phthisis, and some 5 per cent. of cases of phthisis die with pneumothorax. The condition results from the undermining of the pleura by a tubercular focus at the surface of the lung, and it would be much more common than it is were it not for the pleural adhesions which are so nearly constant in phthisis. Fracture of a rib is another frequent cause. Less common causes of pneumothorax are gangrene of the lung, gunshot wounds, rupture of an empyema into the lung or into a bronchus, rupture of an emphysematous bulla, exploratory puncture, perforation of the diaphragm by cancer of the stomach or colon, etc.

Morbid Anatomy.—The opening may be extremely small, or may be large enough to admit a finger-tip. In some cases it is valvular. Its situation is variable. When the perforation takes place, air escapes into the pleural cavity, and sometimes tubercular and purulent matter escapes with it. This causes acute pleurisy, and the effusion is generally purulent, though sometimes serous. The pneumothorax thus gives place to a *pyopneumothorax* (occasionally *hydropneumothorax*). The lung is usually collapsed. If the opening is very small, and no septic matter enters the pleural sac, healing readily takes place by the agency of effused lymph; and after the opening is closed, the air undergoes absorption.

Symptoms.—Perforation as it occurs in phthisis may be marked by the sudden onset of urgent symptoms; but not uncommonly, especially in advanced consumption, symp-

toms are almost entirely absent. Much depends on the question whether the lung so suddenly thrown out of action was the more or less diseased of the two. Among the sudden symptoms are faintness, pain in the chest, dyspnœa or orthopnœa, and the various phenomena of shock. There is sometimes also a feeling that something has given way, and there may be hyperæsthesia of the affected side of the chest.

Physical Signs.—These are characteristic. The affected side of the chest is enlarged and immobile, and the intercostal spaces are obliterated. Vocal fremitus is diminished or absent. Percussion is hyper-resonant or tympanitic. The heart is displaced towards the opposite side. The breath-sound is greatly diminished if not lost, and if it is audible, may be amphoric. After some days, when liquid effusion has taken place, there is dulness in the dependent part and hyper-resonance in the upper part of the affected side. The dulness and resonance readily shift their position with changes of posture.

Amphoric Sounds.—The four important physical signs known as amphoric phenomena may be noted over a pyopneumothorax or a hydropneumothorax, viz., amphoric hum, metallic tinkle, the bell sound and Hippocratic succussion sound (see p. 389). The first three of these signs may be detected before effusion takes place. Succussion may be obtained though there is no other evidence of fluid, the reason being that when pneumothorax sets in, the diaphragm becomes flaccid and even concave above, so that fluid may collect in this concavity without being recognisable by percussion.

Diagnosis.—Three signs are of themselves sufficient, viz., hyper-resonance on the affected side ; displacement of the heart to the opposite side ; and feeble, absent or amphoric breath-sound on the affected side. The history of the case prepares the observer in most instances for the occurrence of pneumothorax. The displacement of the heart distinguishes the case from one of *large intrapulmonary cavity*. A *subphrenic pyopneumothorax* may be suspected from the history.

Prognosis.—The patient generally survives the immediate onset, in which case hectic fever will set in after a day or two, in consequence of the empyema. The pleurisy is due to irritation, not by the air itself, but by the material which escaped along with it into the pleura. If the opening in the pleura is valvular, air is constantly being pumped in by the respiratory act, and its presence at high pressure adds much to the suffering of the patient. Pneumothorax developing in a healthy person is often recovered from, but in the great majority of cases the patient is already suffering from advanced lung disease, and he usually dies within a few weeks. If the pneumothorax occurs on the side which is least affected, death may take place very quickly, but if it causes collapse of the lung which is most diseased, it may not shorten life. It may indeed arrest the pulmonary disease, at least for a time, and the patient may for a considerable period enjoy fair health.

Treatment.—If shock is present at the outset, this must be treated by an opiate. If there is dyspnœa from the presence of air at high pressure within the chest, this must be relieved by puncturing an intercostal space with a fine trocar. Poultices are indicated when the pain is severe. As a rule, no attempt should be made to withdraw the fluid, at any rate for some time. The collapse of the lung may do good by retarding the tubercular process, while attempts to remove the contents of the pleural cavity may reopen the aperture in the pleura which the pleurisy tends to obliterate. In cases where the primary disease is not advancing phthisis, and possibly even in selected cases of early phthisis, the pyopneumothorax may be dealt with by incision and drainage, as if it were an ordinary empyema, provided that sufficient time has been allowed for permanent closure of the aperture in the pleura. In doubtful cases, the safe rule is not to operate.

DISEASES OF THE MEDIASTINUM.

i. Mediastinal Abscess.

Abscess in this situation is not common. Among its causes are blows upon and wounds of the chest-wall, suppuration of the mediastinal lymph glands, erysipelas, pyæmia and other infections, perforation of the gullet, spinal caries, etc. Chronic abscess is often tubercular.

The evidences of abscess are pain, tenderness, hectic fever and pressure phenomena. As the anterior mediastinum is the most common situation, the pain is usually behind the sternum. The pressure phenomena include venous obstruction, paroxysmal cough, dysphagia and dyspnœa. The abscess may point through the chest-wall or may rupture into a bronchus or elsewhere. A chronic abscess may dry in.

The treatment varies according to the case. Poulticing and surgical treatment may be required if the abscess points at the surface. If it communicates with a bronchus, there should be no haste to operate.

ii. Mediastinal Tumours.

Growths in this region are generally malignant. They are most common in middle life, and males suffer more than females. Sarcoma is more frequent than cancer;[1] it is chiefly a lymphosarcoma originating in the glands. Cancer may take origin from the epithelium of the respiratory tract or gullet.

Symptoms.—There may be cough, pain or uneasiness in the chest, and loss of weight, but the diagnosis depends chiefly upon the evidences of pressure on nerves, veins, air-passages and gullet. Pressure on the recurrent nerve—generally the left, on account of its lower situation—may give rise to laryngeal paralysis (recognisable by the laryngoscope, and by changes in the voice and cough) and to attacks of dyspnœa. Pressure on veins applies specially to the

[1] So Lindsay Steven and V. D. Harris, though Hare reverses the order.

superior vena cava, whose tributaries in the head, neck, upper part of the trunk and upper limbs become engorged. In the same way œdema may develop. The subcutaneous veins on the surface of the chest and abdomen become enlarged, and are found to convey the blood downwards to join the tributaries of the inferior cava. Pressure on veins may give rise also to hydrothorax. Pressure on the air-passages and gullet will cause dyspnœa, stridor and dysphagia. Enlarged glands may be found in the neck or axilla. The heart may be displaced. The tumour may give a dull percussion note, and it may erode the chest-wall. Pleural effusion is common and is associated with the usual physical signs.

Diagnosis.—The symptoms may closely resemble those of aneurysm, but are on the whole less paroxysmal and more steadily progressive. The physical signs may point to a tumour at a distance from the large bloodvessels, where aneurysm cannot be looked for. A superficial growth has not expansile pulsation. The patient may be at an age earlier than that at which aneurysm is common, and there may be no sign of arterial disease elsewhere. There is no diastolic shock or tracheal tugging. Enlarged glands in the neck point to tumour. Pressure on venous trunks is in favour of tumour, whereas modifications in the arterial pulse suggest aneurysm. The X rays may reveal an abnormal mass which does not pulsate.

Treatment.—This is purely symptomatic. If fluid accumulates in the pleural cavity, it may be removed as often as is required to relieve the dyspnœa. Opium is indicated for severe pain.

SECTION VI

DISEASES OF THE DIGESTIVE SYSTEM

STOMATITIS.

Inflammation of the mouth is met with in various forms, and may result from irritation by many different agents, such as hot drinks, alcohol, mercury, bacteria and other parasites, broken teeth, etc. The mouth regularly suffers in scurvy.

i. Acute Catarrhal Stomatitis.

This is the most common form of stomatitis. It occurs in infective fevers, in dyspepsia, and as the result of irritation by hot foods, spices, tobacco, etc. It is common at all ages. It is characterised by redness, swelling, and increased sensitiveness of the mucosa, with increase of mucous secretion. There may be discomfort in chewing, but there is little or no constitutional disturbance.

The **treatment** consists in careful cleansing of the mouth with warm water immediately after food, with frequent applications of glycerin of borax. Adults should use a mouth-wash of boric acid, carbolic acid, glycerin and water (glycerini ac. carbol., ℥i.; glycerini ac. boric., ad ℥viii. M. Sig.: A small quantity to be mixed with hot water for the mouth-wash).

ii. Follicular Stomatitis

(Vesicular or Aphthous[1] Stomatitis).

This is most common in the first few years of life, though it is sometimes seen in adults. Vesicles appear on the

[1] See footnote on p. 462.

mucous membrane of the mouth, especially about the lips, cheeks and tongue. These vesicles rupture and give place to little grayish ulcers with red margins. The disease may occur by itself or in association with dyspepsia or an acute fever. Soreness of the mouth, disinclination to take food, increase of secretion and restlessness are the principal symptoms. The ulcers heal within a few days if the dyspepsia or constitutional disturbance is rectified.

Treatment.—The mouth should always be cleansed with warm water after food has been taken, and borax, or glycerin and borax, should then be applied to the mucous membrane. Chlorate of potassium should be given internally in doses of 3 to 5 grains thrice daily for children. In adults the ulcers should be touched with powdered alum or solid nitrate of silver. A good mouth-wash is a 1 per cent. solution of carbolic acid, or a mixture of glycerin of carbolic acid, 1 drachm, with glycerin of borax to 8 ounces, diluted with warm water. Any constitutional disturbance should receive attention.

iii. Ulcerative Stomatitis

(Fœtid Stomatitis. Putrid Sore Mouth).

This disease is not to be regarded as a severe degree of the follicular inflammation just described. It is most common in delicate children under circumstances of defective feeding and hygiene, and is sometimes epidemic, even among adults, in camps and gaols.

After being feverish for some days the child suffers from pain on mastication, salivation, a foul breath, and often swelling of the cheeks, lips and tongue. The affection begins at the margin of the gums, which become red, swollen and ulcerated, and bleed readily. The ulcers are covered with grayish membrane, and in severe cases may penetrate to, and cause necrosis of, the alveolar process. The teeth are loosened, and the lymph glands under the jaw enlarged. The constitutional symptoms may be severe, and death occasionally results. As a rule, however, the ulcers heal in a week or two.

The **treatment** is similar to that recommended for follicular

stomatitis, but the chlorate of potassium should be given in larger doses (10 grains thrice daily for a child; twice as much for an adult). If the breath is very foul, the mouth may be washed with a weak solution of Condy's fluid. Or carbolic and boric acid solution may be employed for cleansing purposes. Abundance of liquid food should be supplied.

iv. Gangrenous Stomatitis (Cancrum Oris. Noma).

This is occasionally seen in very debilitated children, especially girls. A large proportion of the cases occur during convalescence from measles, but it may follow other fevers. The tissues of the cheek become indurated and dark. Ulceration commences on the buccal aspect, and in severe cases proceeds to perforation. The disease sometimes attacks the gums and may extend to the bones. There is little or no pain, but the prostration is great. The pulse is rapid, considerable fever may be present, diarrhœa is common, and aspiration-pneumonia may supervene. Death usually results in about a week.

Treatment is not satisfactory. Feeding and stimulation are important, and the local disease should if possible be destroyed by the cautery or by fuming nitric acid.

v. Parasitic Stomatitis (Thrush).[1]

This variety of stomatitis is due to the development in the epithelial layer of the mucous membrane of a fungus, the *Saccharomyces albicans* or *Oidium albicans*, which consists of a branching mycelium and spores. The disease occurs chiefly in weakly infants, but is occasionally seen in adults in the last stages of an exhausting illness. It may be spread among children by the use of a contaminated feeding-bottle. The fungus gives rise to slightly raised, pearly white patches on the tongue, lips, cheeks and palate, and sometimes the

[1] In the 'Nomenclature of Diseases' (3rd edit., 1896) *aphthæ* is recognised as a synonym for *thrush*, and it is recommended that the use of the term *aphthæ* should be restricted to parasitic disease.

tonsils, pharynx and gullet. The patches are surrounded by a ring of congestion.

The fungus may cause the associated stomatitis, but it is very likely that catarrhal stomatitis, whatever be its cause, will favour the development of the fungus.

According to the depth of the process, the mucosa may appear healthy or ulcerated when the patch is scraped off, but the epithelial layers are seldom completely lost.

The disease may give rise to no definite symptoms, or it may cause discomfort or pain in sucking. There is often concomitant disturbance of the bowels. The disease is distinguished from *follicular stomatitis* by the presence of the fungus, by the dryness of the mouth, and by the absence of the characteristic vesicles and ulcers.

The **treatment** consists in keeping the mouth clean, local applications of glycerin of borax, and correction of errors in the general condition. For the last, a change of air may be highly desirable.

vi. Chronic Stomatitis

(Leukoplakia Buccalis. Buccal Psoriasis).

This condition may set in without obvious cause, or may be due to smoking, spices, alcohol or syphilis. Whitish patches develop on the mucous membrane and may become fissured, but without definite ulceration. The mucosa is abnormally sensitive. The condition sometimes passes on to epithelioma, and should therefore be carefully watched. On the other hand, recovery may take place even after a duration of years.

Treatment.—All causes of irritation (hot drinks, condiments, alcohol, tobacco, etc.) must be avoided. The mouth must be cleansed after the taking of food, and borax in some form may be applied locally.

vii. Geographical Tongue

(Eczema of the Tongue).

This condition is characterised by overgrowth and desquamation of the superficial epithelium of the tongue.

Each focus of desquamation tends to heal at its centre while spreading at its circumference, so that the arrangement is circinate. The fusion of such circles gives rise to wavy tracts. The appearance may thus come to suggest a map or a worm-eaten leaf.

In some cases there are no subjective phenomena, while in others there may be itching, tingling, and heat. The disease is met with in infants and older children, as well as in adults. Nervous patients are apt to fear that it is cancerous, or a manifestation of syphilis. It is sometimes very persistent, and is apt to relapse.

Treatment.—In the case of children, any digestive disorder that is present must be attended to, and an alkaline bitter tonic should be prescribed. In the case of adults, the condition should be treated by the local application of silver nitrate.

DISEASES OF THE SALIVARY GLANDS.

i. Ptyalism.

Ptyalism or excessive secretion of saliva may occur with or without stomatitis. It is sometimes a symptom of pregnancy. It also results from the administration of mercury, the iodides, jaborandi and other drugs, and from the use of tobacco.

ii. Xerostomia.

Xerostomia or dryness of the mouth is apparently the result of neurotic inhibition of the salivary and buccal secretions. There is a general dryness of the mucous membrane of the mouth, and chewing, swallowing and speaking are interfered with. One case mentioned by Osler was cured by galvanism.

iii. Parotitis.

Parotitis occurs in various forms.

(*a*) A *simple parotitis* is described as resulting from morbid conditions in the mouth, such as catarrh, or obstruction of Steno's duct. In some cases there is pain and swelling in

the region of the gland with a certain amount of constitutional disturbance, but there is often no pain.

The cause should be removed, and the duct kept patent if possible.

(*b*) *Specific parotitis* or *mumps* has been described among the infections.

(*c*) *Symptomatic parotitis* (*metastatic parotitis*, *parotid bubo*) is very often suppurative. It occurs in connection with enteric, pneumonia, empyema, pyæmia and other infections, and also as a result of injury or disease of, or operations on abdominal organs. Many if not all of these cases are septic. The infection may reach the gland by the blood, or through the mouth and duct. In most cases the inflammation is confined to one parotid. The fact that post-mortem examinations show that the duct is always affected, and that the gland-lobules suffer before the interstitial tissue, points strongly to the mouth as the source of infection.

Prognosis.—The prognosis is grave if parotitis occurs in the course of a fever, but much less so if it is a sequel.

The **treatment** includes cleansing of the mouth, preservation of the patency of the duct, fomentations locally, and if pus has formed, the usual surgical measures.

DISEASES OF THE TONSILS.

i. Acute Lacunar or Follicular Tonsillitis.

Etiology.—This disease is predisposed to by exposure to cold and wet, an impure atmosphere, rheumatism, gout and general debility, and is probably directly due to microbes. It often occurs in small epidemics.

Symptoms.—At the outset, there are shivering, fever, pains in the head and back, soreness of the throat and pain on swallowing. The tongue is dirty, and the sides of the neck are tender. The tonsils are swollen and red, and abundant yellowish secretion is seen to overflow from the lacunæ. The neighbouring parts may also be inflamed. The disease usually subsides in about five days.

Diagnosis.—It is important not to mistake the discrete yellowish patches of secretion for diphtheritic membrane.

The latter is more continuous, and may be surrounded by much more intense inflammation than is usual in follicular tonsillitis. If the patches are not limited to the tonsils, the case is not simply follicular tonsillitis. In doubtful cases, the bacteriological test should be employed.

Treatment.—The treatment includes rest in bed, saline purges, and abundance of liquid and soft solid food. A mixture containing iron, chlorate of potassium and glycerin may be given internally; or salicylate of sodium may be tried. Gargles are better avoided, as they cause annoyance and do little good.

ii. Chronic Lacunar Tonsillitis.

This variety of tonsillitis is chiefly characterised by excessive production of yellowish secretion by the tonsillar crypts. The tonsils may be enlarged, and the fauces congested. The soft solid secretion when bruised between the fingers gives off an offensive odour.

Treatment.—If the condition gives trouble, the tonsil should be treated by the punch, guillotine or cautery.

iii. Membranous Tonsillitis.

Membranous tonsillitis is sometimes met with apart from diphtheria. It may be due to the pneumococcus or streptococcus, and is sometimes accompanied by widespread inflammatory œdema of the faucial region. After a time, a secondary infection by the diphtheria bacillus may take place. In one case of membranous sore throat which was under my care, the pneumococcus was the cause, and perforation of the fauces supervened.[1] In any doubtful case, a bacteriological investigation should be made.

Treatment.—Isolation is advisable. Rest in bed, abundance of light food, iron with chlorate of potassium internally, and the application of disinfectant sprays are the principal elements in the treatment.

[1] *Glasgow Medical Journal*, 1901, lvi. 274.

iv. Acute Parenchymatous Tonsillitis.

(Cynanche Tonsillaris).

This disease is popularly known as *quinsy*, though the latter designation is more strictly applicable to *peritonsillitis*.

Etiology.—This variety of tonsillitis, like the others, is chiefly seen in adolescents and young adults. It is common in hospital nurses. It may follow exposure to cold or wet, and is sometimes regarded as rheumatic. Thus rheumatism and a liability to sore throat may run in the same family.

Symptoms.—The symptoms resemble those of lacunar tonsillitis, but are more severe. There is great pain and difficulty in swallowing, and the pain may radiate to the ear. Secretion is increased in amount and very tough, so that it is difficult to spit out. The general prostration is considerable. One tonsil usually suffers before the other. The gland is red, tender and swollen ; so much so that it may extend to or beyond the middle line. The neighbouring parts are also swollen. There is tenderness at the angle of the jaw, and the patient can scarcely open his mouth. The temperature may exceed 104° (Fig. 31).

The disease usually subsides after about five days by rapid resolution, but in a good many cases suppuration takes place. This is indicated by aggravation of the pain and tenderness, and sometimes by lividity or other evidence of 'pointing' at one part of the swelling. The *quinsy*, or acute abscess of the faucial region, does not develop in the tonsil itself, but in the connective tissue between the tonsil and the pharyngeal aponeurosis. The quinsy, therefore, is always a *peritonsillitis*, and in nine cases out of ten it forms above and in front of the tonsil. Spontaneous bursting gives great relief. Sometimes an abscess develops in the palate, and contraction of the pupil has been noted as a symptom on the same side as the phlegmon in the velum ; the pupillary change is supposed to be reflex. In rare instances the internal carotid artery has been opened by the suppurative process, or suffocation has resulted from the entrance of pus into the larynx.

Treatment.—A purge should be given early, and salicylate

of sodium may be frequently administered. Gargling should be avoided. The patient may suck ice, or carbolic or guaiac lozenges. Food in liquid or soft solid form should be given at short intervals. If suppuration takes place, hot poultices should be applied to the neck, and the abscess should be opened.

According to StClair Thomson, the site of election is found

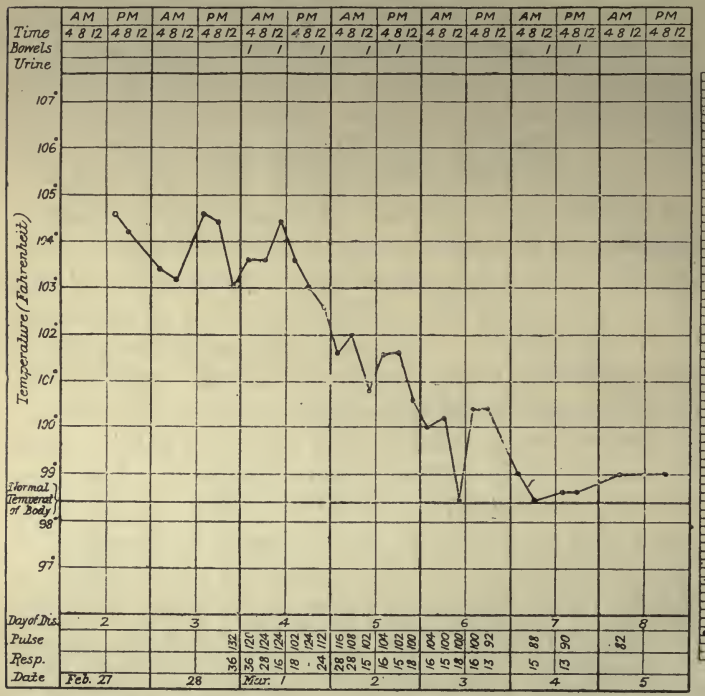

FIG. 31.—ACUTE PARENCHYMATOUS TONSILLITIS, BEGINNING ON BOTH SIDES SIMULTANEOUSLY.

by drawing an imaginary horizontal line across the base of the uvula and a vertical line along the anterior faucial pillar. The point required is immediately external to the intersection of these lines. It is also on a line from the base of the uvula to the upper wisdom tooth of the same side. The part should be cleansed and sprayed with cocaine solution (5 per cent.), and the abscess should be pierced with a pair of Lister's

sinus forceps, the blades of which are to be separated vertically as they are being withdrawn. The boggy feel of the abscess when touched with the forceps indicates the exact site.

In convalescence, tonics are indicated, and a gargle of alum solution should be used several times a day. If attacks recur at short intervals, the tonsils should be cut.

v. Chronic Enlargement of the Tonsils.

Etiology.—This condition is principally seen in children, and tends to recede at puberty. Sometimes it follows a specific fever, or it may follow attacks of the acute lacunar or parenchymatous disease. The enlargement may be due simply to increase of the lymphoid tissue, but sometimes in adults the fibrous tissue is specially increased, with the result that the gland tissue is firm and tough.

Results.—Hypertrophy of the tonsils is often associated with post-nasal adenoids, which are due to hypertrophy of the lymphatic tissue of the naso-pharynx. Both are apt to be associated with chronic or recurring nasal catarrh. The tonsillar enlargement does not necessarily cause any trouble beyond a special liability to acute tonsillitis and naso-pharyngeal catarrh, but along with post-nasal adenoids it is apt to give rise to nasal obstruction and mouth-breathing. These various conditions tend, singly or collectively, to cause changes in the quality of the voice, in the shape of the chest (pigeon-breast), and in the facial expression ; and also to cause deafness, cough, night terrors and asthma.

Treatment.—Though the tonsils tend to shrink at puberty, this epoch must not be waited for if there is any appearance of such symptoms as deafness and mouth-breathing. The enlarged tonsil should be removed by the guillotine, or by some other means. The naso-pharynx should at the same time be examined for adenoids and treated in the appropriate manner. In tubercular children, cod-liver oil and iron, with a change to the seaside, may be beneficial after operations on this region.

DISEASES OF THE PHARYNX.

i. Acute Pharyngitis.

Etiology.—This may be associated with severe diseases, such as tonsillitis, diphtheria and scarlet fever, but it also occurs as a simple catarrh (*catarrhal sore throat*), resulting from exposure, rheumatism, or the action of more obvious irritants (hot drinks, tobacco-smoke, etc.). The fauces and nares often suffer simultaneously.

Symptoms.—There is a sense of dryness or rawness about the throat. The secretion is increased, and after a time becomes purulent. The affected parts are congested and dry, and hearing may be impaired.

Treatment.—Little treatment is required. An atmosphere of uniform temperature, a dose of Dover's powder at bedtime, and a laxative are sufficient. Menthol jujubes may be sucked. If the condition tends to recur frequently, the nose should be examined, and if need be, treated.

ii. Chronic Pharyngitis.

Etiology.—This disease may result from repeated acute attacks, or from constant smoking, spirit drinking, or excessive use of the voice (whence the name *clergyman's sore throat*). It is often associated with chronic catarrh of the naso-pharynx.

Symptoms.—The mucous membrane is red and relaxed. Small, superficial ulcers may be present. In some cases, red nodules are seen on the posterior wall of the pharynx, resulting from overgrowth of lymphoid tissue (*granular* or *hypertrophic pharyngitis*). In other instances the mucosa is thin, smooth and dry (*dry* or *atrophic pharyngitis*).

Chronic pharyngitis may cause no trouble, or there may be local discomfort with impairment of voice, and a frequent desire to clear the throat.

Treatment.—The cause should be removed if possible. Change of air and tonics may be helpful. If the symptoms continue, the granulations should be cauterised.

iii. Retropharyngeal Abscess.

Retropharyngeal abscess may be acute or chronic. The *acute* variety occurs chiefly in the first two years of life, and is due to suppuration of lymphatic glands which lie between the pharynx and the cervical vertebræ. The symptoms include fever, pain on swallowing, dyspnœa and cyanosis. The posterior wall of the pharynx can be seen or felt to bulge forwards.

The *chronic* abscess occurs in older children, and is generally due to tuberculosis of the cervical vertebræ, of which other evidence is usually present. There are few or no constitutional symptoms.

Treatment is surgical.

iv. Angina Ludovici

(Ludwig's Angina. Submaxillary Cellulitis).

Etiology.—This condition is the result of septic infection of the cellular tissues in the submaxillary region. It may be idiopathic, traumatic, or secondary to specific fevers. A streptococcus is the usual microbe.

Symptoms.—There are induration, tenderness and heat in the affected region, and there is danger of pressure on important structures, extension to the larynx, and systemic infection.

Treatment.—Prompt surgical measures are necessary.

DISEASES OF THE ŒSOPHAGUS.

i. Œsophagitis.

Œsophagitis may be acute or chronic.

Acute inflammation results from the swallowing of boiling liquids, strong acids or alkalies, or from wounds due to foreign bodies. It also occurs spontaneously in infants. The principal symptom is pain on swallowing. There may be tenderness over the gullet. In severe cases, rectal feeding may be necessary.

Chronic inflammation may follow the acute disease. It is commonly present above any stricture of the tube.

ii. Ulceration of the Œsophagus.

Ulceration may be catarrhal or malignant. At the lower end of the œsophagus an ulcer is occasionally found similar to the simple perforating ulcer of the stomach.

iii. Varicose Veins in the Œsophagus.

Varicose Veins may be very marked at the lower end of the gullet in cases of cirrhosis of the liver with portal obstruction. Rupture of such a varix is one mode of death in hepatic cirrhosis.

iv. Obstruction of the Œsophagus.

Etiology.—Obstruction of the œsophagus may be due to foreign bodies in the lumen (*e.g.*, false teeth or a piece of bone); muscular spasm; structural changes in the wall of the tube (*e.g.*, a malignant growth or cicatricial stricture); or compression from without (*e.g.*, by aneurysms or other mediastinal tumours).

Spasmodic Stricture (*œsophagismus*) occurs in neurotic subjects, both male and female. It is chiefly met with in early adult life. The spasm is recognised by a difficulty in swallowing, which is sometimes accompanied by pain or discomfort in the chest. The obstruction may yield to a bougie at once, or only after continuous gentle pressure has been applied to it for a time.

Diagnosis.—The sudden onset of the spasm in a young adult, the possibility of passing a large bougie, the absence of other symptoms, and the neurotic disposition of the patient will permit of the diagnosis being made.

Treatment.—The passage of a large bougie now and then may effect a cure, but it is important to treat the general condition.

Cicatricial Stricture (*fibrous stricture*) is usually situated either at the upper or at the lower end of the gullet. It is generally due to the swallowing of some corrosive material, though when situated at the lower end its cause may be the healing of a simple ulcer.

Symptoms.—The principal symptom is a gradually increasing difficulty in swallowing, at first of solids, but ultimately also of liquids. If the obstruction is high, food and drink are returned immediately ; if it is low down, the gullet becomes dilated above the stricture, and the food is later in being rejected. There is little or no pain, though the patient can indicate the level of the obstruction. The tendency is towards death by gradual starvation. The history is often a valuable guide in diagnosis. Before a bougie is passed, it is necessary to examine the chest carefully to exclude aneurysm.

Treatment.—The treatment consists in gradual dilatation of the stricture by bougies of increasing size. If this method is impracticable, gastrostomy must be performed.

v. Cancer of the Œsophagus.

(Malignant Stricture).

Etiology.—The disease occurs chiefly in the second half of life, and is more common in men than in women.

Morbid Anatomy.—Cancer of the gullet is almost always a flat-celled epithelioma which begins in the mucous membrane, and tends to spread round the tube. It measures 1 or 2 inches longitudinally. It projects into the lumen and usually undergoes ulceration. It is met with chiefly in the lower and middle thirds, and is especially frequent at the level of the bifurcation of the trachea (opposite the fourth dorsal spine). It often involves the lymph glands in the chest and neck, and may extend to the respiratory passages, lung, pleura, and aorta.

Symptoms.—The most important early symptom is increasing dysphagia, first as regards solids, and later with regard to liquids also. The result is that before long rapid loss of flesh and strength takes place. Ulceration of the stricture may lead to temporary improvement. Pain may be present continuously, or only on swallowing, but is usually slight or altogether absent. The food that is rejected may be accompanied by blood. The cervical lymph glands may be enlarged, and the recurrent nerves may be

involved. Perforation into the respiratory passages, lung, pleura, aorta, etc., leads naturally to formidable complications. Death usually takes place within a year from exhaustion, or from broncho-pneumonia, pulmonary gangrene, hæmatemesis, or some other result of perforation.

Diagnosis.—The age of the patient and the progressive character of the obstruction are very significant. Recent enlargement of cervical glands points in the same direction. It is important to exclude aneurysm by the symptoms and physical signs, and to exclude cicatricial stricture by the history. The level of the obstruction is determined by the patient's feelings, and more accurately by the distance to which a sound can be passed. (From the incisor teeth to the cardiac orifice is normally 16 inches.)

Prognosis.—This is hopeless, though life may be prolonged in some cases by gastrostomy.

Treatment.—The food swallowed may be supplemented by rectal feeding. Sometimes a tube is kept permanently in the œsophagus to allow of regular feeding. The growth should not be irritated by repeated attempts to pass bougies. It is much better to perform gastrostomy as soon as the patient becomes unable to take a sufficiency of food.

vi. Dilatation of the Œsophagus.

Dilatation of the gullet takes place above any permanent obstruction, and is associated with hypertrophy. Primary dilatation is very rare. In either case, dysphagia and regurgitation of food are the principal symptoms.

vii. Diverticula.

Diverticula are due either to (*a*) pressure, or to (*b*) traction.

(*a*) The *pressure diverticulum*, or *pressure pouch*, is most common at the back of the gullet at its upper end. Owing to weakness of the muscular wall at this part, the mucous and submucous layers bulge backwards between the muscular fibres, and the accumulation of food causes gradual enlargement of the sac. An important symptom is regurgitation of undigested food many hours, or even a day or two, after it has been taken. Acidity of such material is

no evidence against the existence of a pressure pouch. Another symptom is gurgling up of gas from the throat, especially when pressure is applied low down on the left side of the neck. In the third place, a bougie is arrested about 9 inches from the teeth. Occasionally there is bulging in the neck at the region of the pouch. In an early stage it may be possible to pass a large bougie into the stomach. The presence of a large pouch may be associated with emaciation, and with pressure-symptoms, such as cough. The most satisfactory treatment is excision of the diverticulum.

(*b*) The *traction diverticulum* involves the front of the œsophagus, at the level of the bifurcation of the trachea. The anterior wall of the gullet, having become adherent to some neighbouring structure, such as a diseased lymph gland, is drawn forwards by cicatricial contraction. No symptoms result, unless a foreign body lodges in the sac.

DISEASES OF THE STOMACH.

i. Methods of Examination.

The long axis of the stomach is more nearly vertical than was until recently supposed. The cardiac orifice corresponds to the seventh left costal cartilage, while the pylorus is 2 or 3 inches below the ensiform cartilage and about 1 inch to the right of the middle line. The healthy stomach usually yields a tympanitic note over a crescent-shaped area (Gairdner's *gastric crescent*) immediately below the left lower costal margin, and this note may be obtained over the lower ribs as far up as the fifth left intercostal space external to the apex beat of the heart (Traube's *semilunar space*). Unless the healthy stomach is greatly distended by food and drink, it cannot be recognised by inspection or palpation of the epigastrium. The peristaltic movements are not seen. It has also been asserted that succussion gives rise to no splashing sound, but such a sound can undoubtedly be obtained in this region, whether it be in the stomach or in the colon, in perfectly healthy persons.

When the organ is dilated, it may give rise to a visible prominence of the epigastrium, and it may then be accessible also to palpation. Peristaltic movements are observed, passing very slowly across the abdomen, usually from left to right, and capable of being excited by friction over the stomach. Shaking the patient's abdomen produces a splashing sound if the stomach contains much liquid. The area over which the stomach note is obtained by percussion is much increased, and is best mapped out by *auscultatory percussion*. To carry out this method, the observer listens with the stethoscope over what is undoubtedly stomach—for instance, in Traube's space—while he carefully notes the sound produced by percussion close beside the stethoscope. He then proceeds to percuss at a distant part of the abdomen, and gradually moves the pleximeter towards the stethoscope, until the percussion note becomes identical with that already heard on percussion over the stomach; this phenomenon indicates that the pleximeter has reached the area of the dilated stomach.

Some of these methods of examination are facilitated if the organ has been previously distended by carbon dioxide. For this purpose the patient drinks two solutions, one after the other; the one containing ½ a drachm or a drachm of tartaric or citric acid in 2 ounces of water, and the other containing a similar quantity of sodium bicarbonate. The vertical extent of gastric resonance should not exceed 5 or 6 inches in the male, or 4 inches in the female.

In health, the *capacity* of the organ is less than 3 pints. In disease, it may be estimated from the large quantities of material which a patient will vomit at one time, or by introducing into the stomach by the tube as much warm water as the patient can comfortably tolerate, and then removing the total contents for measurement.

Examination of the vomited matter often gives information of great value, and in many cases it is desirable to remove the contents of the stomach artificially for the same purpose. With this object in view, it is a common practice to give the patient a *test meal*, and then to

withdraw the stomach contents after an interval of one or more hours. The test breakfast of Ewald and Boas consists of bread (½ ounce to 3 ounces) and weak tea (10 to 13 ounces); and the contents are examined after one hour. Klemperer recommends 1 pint of milk instead of the tea, but the curdled masses of milk are apt to obstruct the eyes of the tube. Other meals contain more proteid, and therefore test more thoroughly the special digestive capacity of the stomach. Thus, Germain-Sée gives bread (3 to 5 ounces), minced meat (2 to 2½ ounces), and water (a tumblerful); the stomach contents are examined about two hours afterwards. Herschell's test breakfast consists of a lightly boiled egg, minced meat (1 ounce), toast (3 ounces), and ¼ of a pint of very weak tea; the contents are examined an hour and a half later. Leube and Riegel give a test dinner of soup (13 ounces), scraped beef (2 ounces), and bread (1¾ ounces); in this meal there is so much proteid that from four to six hours should be allowed to pass before the examination is made. In actual practice, however, it is perhaps as well to let the patient take what is for him an ordinary meal, and to examine the stomach contents after an interval of time which may be judged to correspond to the digestibility of the ingredients of the meal.

The stomach contents having been removed by the tube, their *reaction* is demonstrable by litmus-paper; it is almost invariably acid. Their *total acidity* (which is due to free acids, acid salts, and acids combined with proteids) is estimated by means of a decinormal[1] solution of sodium hydrate (4 grammes in 1,000 c.c. of distilled water) and

[1] A *normal solution* of an acid is such that a litre of the solution contains 1 gramme of replaceable hydrogen. Therefore, a litre of normal solution of an acid which contains one atom of replaceable hydrogen will contain as many grammes of the acid as are represented by its molecular weight (*e.g.*, HCl: molecular weight, 36·5; normal solution, 36·5 grammes per litre); while a litre of normal solution of a bibasic acid will contain half as many grammes as correspond to its molecular weight (*e.g.*, H_2SO_4: molecular weight, 98; normal solution, 49 grammes per litre). Similarly a normal solution of caustic soda is such that the number of grammes in a litre of the solution is the same as the molecular weight of the soda, viz., 40 (Na=23, H=1, O=16; NaHO=40). It will be noted that

an alcoholic solution of phenolphthalein (1 per cent.). Phenolphthalein is an aniline body which gives a slightly yellow solution when dissolved in alcohol. It is used to indicate when the acid reaction disappears from the fluid which is being examined. Ten c.c. of the stomach contents are placed in a beaker. A few drops of the phenolphthalein solution are added to this, the mixture remaining almost colourless. The decinormal sodium hydrate solution is now added drop by drop from a burette, the mixture being at the same time continually stirred, until the appearance of a pink colour shows that the contents of the beaker are no longer acid. The acidity is stated in terms of the amount of the decinormal solution required to neutralise 100 c.c. of the stomach contents. *E.g.*, if 5·3 c.c. of the decinormal solution in the burette had to be added to the 10 c.c. of stomach contents in the beaker before the change of colour took place, the 'degree of acidity' would be reckoned as 53. The degree of acidity of normal gastric contents is between 40 and 60. The total acidity can also be expressed in terms of hydrochloric acid; 40 grammes of NaHO being neutralisable by 36·5 grammes of HCl (H = 1, Cl = 35·5). *E.g.*, if 5 c.c. of the decinormal solution must be added to the beaker to neutralise the 10 c.c. of stomach contents, it would require 500 c.c. of the decinormal solution, or 50 c.c. of a normal solution of NaHO, to neutralise 1,000 c.c. (1 litre) of the stomach contents. Fifty c.c. is $\frac{1}{20}$ of 1 litre, and $\frac{1}{20}$ of 1 litre of normal NaHO, solution (containing $\frac{1}{20}$ of 40 grammes, viz., 2 grammes) is molecularly equivalent to, and neutralisable by, $\frac{1}{20}$ of a litre of normal HCl solution (containing $\frac{1}{20}$ of 36·5 grammes, viz., 1·8 grammes). If, therefore, 1,000 c.c. of the stomach contents contained 1·8 grammes of hydrochloric acid, 100 c.c. would contain 0·18 gramme of hydrochloric acid. In other words, the

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a normal solution of sodium chloride (Na = 23, Cl = 35·5; NaCl = 58·5) has a strength of about 60 per 1,000, or 6 per cent., viz., ten times the strength of what has long been known and used clinically under the name of 'normal saline solution.' The latter ought to be called *physiological saline solution;* it is now known that its strength for human beings should be about 0·9 per cent.

acidity of the stomach contents in terms of hydrochloric acid was 0·18 per cent. Under normal circumstances it is about 0·2 per cent.

If *free acids* are present, or even a large amount of combined hydrochloric acid, Congo red test-paper is rendered blue. Acid phosphates do not cause this reaction. Congo red solution is more sensitive than the test-paper. One or two drops of the watery solution (1 in 1,000) may be added to 5 or 10 c.c. of the filtered gastric contents, whereupon a blue colour develops if free acids are present.

Another mode of testing for free acids is by placing some of the gastric contents in a test-tube and adding calcium carbonate. If effervescence takes place, free acid is present. If, after filtering, the fluid still retains its acidity, acid phosphates are present, since free acids are neutralised by calcium carbonate.

Congo red paper which has been rendered blue by free acid is deprived of this blue colour by ether in the case of organic acids, but not in the case of mineral acids.

Free hydrochloric acid is recognised by a colour reaction with Günzburg's or Boas's reagent. Small quantities of the stomach contents (filtered or unfiltered) and of the reagent are mixed in a porcelain capsule, and caused to evaporate by gentle heating. If free hydrochloric acid is present, a rose-red colour is produced. Günzburg's reagent consists of phloroglucin 2 grammes, vanillin 1 gramme, and absolute alcohol 30 c.c. Boas's reagent consists of resorcin 5 grammes, white sugar 3 grammes, and weak spirit 100 c.c. A still more delicate test for free hydrochloric acid is by dimethyl-amido-azobenzol, which is used in the form of a 0·5 per cent. alcoholic solution. From 5 to 20 c.c. of filtered or unfiltered stomach contents are placed in a test-tube, and to these there are added, by means of a pipette, from 1 to 3 drops of the dimethyl-amido-azobenzol solution. If free hydrochloric acid is present, a cherry-red colour is produced, the intensity of which varies in proportion to the amount of the free acid.

Lactic acid is a fixed acid, and is best detected after extraction with ether, which is shaken up with the gastric

contents, allowed to separate, and then decanted off. The ether is then evaporated, and the residue is dissolved in water. If lactic acid is added to an almost colourless solution of perchloride of iron, the latter becomes canary yellow. A delicate test is that of Uffelmann, whereby a drop or two of solution of ferric chloride is added to, say, 30 c.c. of a 2 per cent. solution of carbolic acid, with the result that an amethyst blue is produced. The addition of a trace of lactic acid turns the blue into yellow.

Butyric acid is volatile, and may be recognised by its odour, which resembles that of rancid butter. If a little alcohol and sulphuric acid be added to the liquid in which it is contained, and the mixture be then heated, the characteristic odour of butyric ether is evolved. Or 10 c.c. of the gastric contents may be mixed with 50 c.c. of ether, and evaporated to dryness. To the residue a few c.c. of water are added, and also a trace of calcium chloride. If butyric acid is present, it will be precipitated in the form of small oily-looking odoriferous globules.

Acetic acid is also volatile, and recognisable by its odour. It is not so important in gastric disease as lactic and butyric acids. It gives a blood-red colour with perchloride of iron. The test may be carried out by mixing 10 c.c. of filtered stomach contents with 50 c.c. of ether, evaporating to dryness, dissolving the residue in a little water, and cautiously neutralising with a weak solution of sodium hydrate. If acetic acid is present, sodium acetate is formed, and when two or three drops of a weak solution of ferric chloride are added to the mixture, a dark red colour is produced.

The *motor efficiency* of the stomach may be tested in various ways. Thus (1) a meal containing soup, beef and bread should have disappeared from the stomach (as shown by the tube) within six hours, or, at any rate, nothing but minute particles of food should be recognisable. Or (2) 15 grains of salol may be given during the digestion of a meal. When the salol reaches the duodenum, it is decomposed by the alkaline contents into its constituents, phenol and salicylic acid, and the latter being absorbed, becomes recognisable in the urine by giving a violet colour with ferric chloride solu-

tion. A drop of urine and of the iron solution should be allowed to run together on a porcelain plate or on filter-paper. The reaction ought to appear in about an hour after the salol is taken, and to disappear after twenty-four hours. Or again (3) ½ a drachm of iodipin (iodine chemically combined with sesame oil) may be given in capsule at breakfast. The iodine is not liberated till it reaches the intestine, where it is absorbed. The saliva is tested with starch paper every few minutes till the iodine reaction appears, which ought to be in from fifteen to forty-five minutes.

The *absorptive power* of the stomach may be tested by giving 3 grains of potassium iodide in a capsule with a drink of water. The iodide is absorbed from the stomach, and reaches the blood. Every few minutes starch paper is applied to the tongue, and then fuming nitric acid is allowed to drop on the moistened paper so as to liberate the iodine. The iodide of starch reaction (blue colour) usually appears within fifteen minutes.

ii. Symptomatology.

Dyspepsia or *indigestion* in the sense of imperfect gastric digestion is one of the most common ailments, and results from many disorders of the stomach, both functional and structural, which will be described hereafter. The symptoms which result are very various, and certain combinations of symptoms are specially characteristic of particular forms of gastric disease. Some of the symptoms are due to fermentative changes in indigestible or at least undigested food. The products may be gaseous and cause *flatulent distension ;* or they may cause *pain* by directly irritating the mucous membrane ; or again they may excite catarrh and thus cause, not only pain, but the various other symptoms of gastritis. Pain may be due to irritation of an ulcer by food or by gastric juice, or it may be due to a new growth. The pain may radiate from the ' pit of the stomach ' (*scrobiculus cordis*), round the left side, or through the trunk to the back. Sometimes it is reflected along intercostal nerves to the back of the chest about the lower angle of the scapula. *Tenderness* is often associated with pain, but in neuralgic

conditions, pain may be relieved by pressure as well as by the taking of food. Pain in the gastric region is sometimes called *gastrodynia*. When it is localised near the heart, it is called *heartburn* or *cardialgia*.

Nausea and *vomiting* are common in severe gastric disturbance. If vomiting is frequently repeated, bile often regurgitates from the duodenum into the stomach and is vomited. Vomiting of blood (*hæmatemesis*) is an important symptom of simple and malignant ulcers and of hepatic cirrhosis. It may also occur in purpura. When blood oozes into the stomach in small quantity, the hæmoglobin is changed by the gastric juice into hæmatin, and the vomited matter has the appearance of coffee grounds. *Waterbrash* (*pyrosis*) means the eructation of acid fluid from the stomach accompanied by burning pain in the epigastrium, but the term is often applied to the regurgitation of a non-irritating fluid which is perhaps saliva that has been swallowed.

The condition of the *tongue* is very often altered in connection with gastric disturbance, especially in the way of increase or diminution of the normal fur on its dorsum. *Disturbances of the heart's action* are also common in dyspepsia, partly because flatulent distension of the stomach causes upward pressure upon the heart, and partly through disturbed innervation resulting from absorption of substances taken into, or formed in the stomach. That the *nervous system* also suffers is shown by the headache and mental depression which are so common, and which probably are due to harmful agents generated in the stomach and not completely destroyed as they pass through the liver.

iii. Hæmorrhage from the Stomach (Gastrorrhagia).

Hæmorrhage into the stomach is known as *gastrorrhagia*. If the blood is vomited, it is *hæmatemesis*.

Etiology.—The bleeding may be due to rupture of dilated veins, or to simple ulcer, cancer, acute or chronic catarrh, passive hyperæmia, certain fevers (*e.g.*, small-pox and yellow fever), phosphorus poisoning, hæmophilia, purpura,

or profound anæmia. It may occur in nervous conditions such as hysteria, and it may vicariously replace menstruation. It may follow injuries of the epigastrium or the swallowing of corrosive poisons. It may follow operations on the abdomen—*e.g.*, for appendicitis.

It is to be noted that blood which is vomited is not necessarily derived from the stomach in the first place. It may have come from the mouth, nose, or respiratory organs, and have been swallowed; or it may come from an aortic aneurysm bulging into the œsophagus, or from a varicose vein at the lower end of the œsophagus. Patients are apt to speak of blood being vomited when it comes up into the mouth in considerable quantity, even though it may come from the respiratory tract. Blood from the air-passages is often brought up by coughing; it is bright red, frothy, and alkaline; in many cases there are symptoms and signs of pulmonary, cardiac, or aneurysmal disease; and the sputum may contain blood for some time afterwards. On the other hand, blood from the stomach is brought up by vomiting; it is dark, non-frothy, and often acid; gastric symptoms are commonly present; and melæna follows the attack. The feelings of the patient may indicate the seat of hæmorrhage.

Passive hyperæmia of the stomach is caused by obstruction to the portal circulation (*e.g.*, by cirrhosis of the liver, thrombosis in the portal vein, compression of the vein by growths); it sometimes occurs in association with splenic enlargements; and it is met with as part of the general passive congestion of heart disease and emphysema.

The most common causes of hæmatemesis are ulcer of the stomach, especially in women; cancer of the stomach, in either sex; and cirrhosis of the liver, especially in men.

Morbid Anatomy.—If death was due to the hæmorrhage, there are signs of profound anæmia. In cases of ulcer and cancer, the source of the hæmorrhage is manifest. In cirrhosis of the liver there are often varices in the œsophagus, and one of these, by rupturing, may be the cause of the bleeding. But the mucous membrane of the stomach itself may show no sign of lesion, or it may present a pinhole erosion leading down to a ruptured vein or miliary aneurysm

In toxic cases (fevers, phosphorus poisoning, etc.) hæmorrhage is found in the mucosa.

Symptoms.—In rare cases death occurs before vomiting takes place. In other severe cases the well-known symptoms of hæmorrhage present themselves, including pallor, faintness, etc. In cases of ulcer, hepatic cirrhosis, and splenic enlargement, the bleeding may prove fatal; but this, particularly in the case of ulcer, is exceptional. Patients with ulcer may suffer during a period of years from recurring attacks of hæmatemesis.

iv. Acute Gastric Catarrh

(Acute Gastritis. Acute Dyspepsia).

Etiology.—Acute gastric catarrh occurs at all ages and in both sexes. Some persons are much more liable to suffer than others. The usual cause is irritation of the stomach by food which is unsuitable in kind and excessive in quantity. The surplus which the stomach cannot digest undergoes decomposition, and the products of this process irritate the mucous membrane. Babies are apt to suffer through the use of unclean feeding-bottles. Children may suffer from unripe or overripe fruit. A drinking bout is a common cause in adults. In some people a chill seems to cause gastritis along with bronchitis. In some of the infectious fevers there may be acute catarrh of the stomach.

Morbid Anatomy.—The mucous membrane is red, swollen and covered with mucus. It is sometimes the seat of hæmorrhages. The epithelial cells are swollen and more granular than in health. The gastric juice is diminished in quantity.

Symptoms.—These correspond to what is popularly known as a 'bilious attack.' Since the fermentation products absorbed from the stomach must pass to the liver by the portal vein, it is intelligible that the hepatic functions may be disturbed, as is implied by the expression 'biliousness.' The symptoms include discomfort, tenderness and occasionally severe pain in the region of the stomach, with headache, loss of appetite, a bad taste in the mouth, sometimes sickness and vomiting, and a furred tongue. The vomited matter may be found to contain no hydrochloric acid, but

an excess of mucus, and very likely fatty (butyric and acetic) and lactic acids. In severe cases, there may be fever at the outset.[1] The urine often deposits urates on standing. Constipation is the rule, but there is often diarrhœa, especially in children, from concomitant enteritis. The attack usually passes off in a few days.

Diagnosis.—This is seldom difficult, but a day or two may elapse before it is possible to exclude the *infectious fevers*. The *gastric crises* which are sometimes an early or even initial symptom of locomotor ataxy must not be mistaken for gastric catarrh, and the importance of vomiting as a symptom of *intracranial disease* must never be forgotten. In *acute intestinal obstruction*, the constipation is frequently absolute, so that not even flatus is passed; the pain becomes intense, with frequent exacerbations; the vomiting also becomes more and more distressing, and tends to become fæcal in character; while the constitutional symptoms are of a grave type.

Treatment.—Abernethy defined biliousness as a condition curable by blue pill, and there is no better medicine at the outset than mercury in some form. This may be accompanied or followed by a laxative pill or draught if there is constipation, and should be accompanied by bismuth if there is diarrhœa or vomiting. If there is much gastric pain and retching, it is well, at the outset and in the case of adults, to give a large quantity of warm water so as to induce vomiting and thus wash out the stomach. If the pain still continues severe, frequent hot fomentations should be applied locally, and a dose of morphine may be given hypodermically. The stomach must have as much rest as possible, and the patient should live for a day or two on milk diluted with soda-water or barley-water. Complete rest in bed till all gastric disturbance passes off is highly desirable.

v. Phlegmonous Gastritis
(Acute Suppurative Gastritis).

In this rare disease, inflammation of the stomach wall goes on to suppuration, usually in the submucous layer, and either diffusely or in the form of a definite abscess. It may be idiopathic or pyæmic.

[1] 'Gastric fever' is really a mild form of enteric fever.

Symptoms.— The symptoms are great pain and tenderness in the region of the stomach, anorexia and vomiting, with fever and other symptoms of septic poisoning leading to a fatal issue. The abscess can sometimes be felt externally, and if it ruptures into the stomach, pus may be vomited; but the diagnosis is difficult. Recovery may occur if the abscess ruptures into the stomach.

Treatment.—Unless the swelling can be felt, treatment must be symptomatic.

vi. Toxic Gastritis.

This form of gastritis is caused by the swallowing of corrosive or irritant poisons, such as mineral acids, caustic alkalies, oxalic acid, carbolic acid, arsenic, corrosive sublimate, potassium chlorate and potassium cyanide. Phosphorus causes fatty degeneration of the gastric glands and muscle, but the other poisons mentioned cause in some parts necrosis of tissue and in others intense inflammation, the latter being manifested by redness, swelling and hæmorrhage. If the patient survives, ulcers are thus produced in the most affected areas, while chronic inflammation leading to fibrosis ensues in the less affected portions.

Symptoms.—The symptoms include intense gastric pain and tenderness, vomiting, and in severe cases shock which is often fatal. If the patient survive, he is likely to suffer from chronic gastric symptoms. There will probably be pain in the mouth and throat, salivation, and dysphagia.

Diagnosis.—The history of swallowing the poison, the sudden onset of severe symptoms in the midst of good health, the condition of the mouth and throat, and the character of the vomited material ought to give the clue to the diagnosis.

Treatment.—This includes the administration of an antidote for the poison, and morphine to relieve pain. Otherwise it is symptomatic.

vii. Membranous Gastritis.

This condition is occasionally observed in connection with infectious diseases such as pneumonia, pyæmia, typhus,

enteric, etc. It is seldom met with in diphtheria. The symptoms are not distinctive, but it may occasionally happen that a piece of membrane is vomited.

viii. Chronic Gastric Catarrh

(Chronic Gastritis. Chronic Dyspepsia).

Etiology.—This condition may follow acute gastritis, or may follow the prolonged operation of agents which cause slight irritation of the mucosa. Thus, habitual over-eating, excessive use of tea, alcohol and iced drinks, irregularity of meals, and imperfect mastication are causes. Morbid conditions of the stomach, such as cancer, dilatation and passive hyperæmia, lead to chronic inflammation. Conditions associated with general debility, such as chlorosis, phthisis, and overwork, lead to loss of tone of the gastric muscle and favour indigestion. Nervous states such as anxiety, which temporarily inhibit the gastric functions, may cause inflammation by permitting undigested food to remain in the stomach.

Morbid Anatomy.—This varies greatly in different cases. In the early stages, the inflammation may be parenchymatous as well as interstitial, but in the long-run it tends to produce an increase of interstitial and destruction of glandular elements. In many cases the organ is enlarged. The wall may be thickened or thinned. The mucous membrane may be dark from old-standing congestion and hæmorrhages. Little ulcers, from which serious hæmorrhage may take place, are sometimes met with (*hæmorrhagic erosions*). The mucosa may be thinned, or it may be *mammillated*—namely, elevated into little nipple-like prominences (*gastritis polyposa*).

Occasionally in advanced cases the stomach is much reduced in size, while its wall is greatly thickened (*cirrhosis ventriculi*);[1] or, again, there may be thinning of the wall

[1] Some of these cases closely resemble, if indeed they are not, *diffuse scirrhous infiltration* of the stomach wall (*indiarubber-bottle stomach*). In the latter condition there is a tendency for the epithelial cells to disappear from the alveoli of the cancer, but under such circumstances they have been discovered in the neighbouring lymph glands.

with preservation or increase of the size (*phthisis ventriculi*).

Symptoms.—There may be slight epigastric tenderness. Pain in the stomach is common, and is apt to be aggravated an hour or two after meals. Vomiting may give some relief. A 'stomach pain' is often felt also at the back of the chest, at the lower scapular or interscapular region. The appetite is variable, the tongue is furred, and there is a bad taste in the mouth. Nausea and vomiting are frequent, and habitual drinkers in particular vomit in the mornings. The vomited matter contains food in various stages of digestion, mucus, lactic acid, and sometimes butyric and acetic acids; but hydrochloric acid is deficient. The stomach is often distended with gas, so that the patient may have to loosen the clothing shortly after meals. Gaseous eructations may be troublesome. The tests for gastric digestion and absorption show that these processes are delayed. The bowels are usually costive, but may be irregular. The urine often deposits urates on standing. Headache, giddiness, and low spirits are well-known symptoms.

Diagnosis.—The history of the case and the character of the symptoms are usually sufficient, but sometimes it is desirable to carry out the various procedures mentioned in the section on examination of the stomach. Even then it may sometimes be necessary to watch the progress of the case for some time before certainty can be attained. Atrophy of the gastric mucosa may, like cancer of the stomach, give rise to symptoms very similar to those of pernicious anæmia.

Prognosis.—If the cause of the disease can be removed, great improvement, and even a practical cure, may be expected.

Treatment.—The patient must set aside all improper dietetic habits, such as over-eating, over-drinking, irregularity of meals, imperfect mastication, and excessive work of muscle or brain immediately after dinner. The diet for a time must be much restricted. It is better that the patient should take a small quantity of food and digest it, than take much and fail to digest it. The undigested

portion is not simply useless and wasted, and a needless burden to the disabled stomach, but by undergoing fermentation it becomes positively hurtful. It may be desirable for a time to wash out the stomach every day or every second day.

Tea, coffee, and alcohol should be avoided, and replaced by milk and cocoa. Pork, green vegetables, pastries and fatty and sweet articles should also be avoided for some time. Beginning with milk and milk foods, the patient may gradually add to his diet boiled white fish, boiled chicken, the lean of mutton and beef, small quantities of mashed potato, pea-soup, pease porridge, and oat-flour porridge. The bowels should be opened daily, if need be by the aid of cascara, or of aloes with iron, at night, or by a saline in the morning. A small dose of mercury once or twice a week will be advantageous. An alkaline bitter tonic with nux vomica may be given before meals to stimulate the secretion of the gastric juice and improve the tone of the gastric muscle ; or, again, dilute hydrochloric acid may be given, with or without pepsin, just after meals. Bismuth may be given for the relief of pain, and soda-mint or charcoal after meals for flatulence, acidity, or heartburn. If these symptoms prove obstinate, the stomach should be washed out. Dilute hydrocyanic acid with bismuth is indicated for vomiting, and this symptom, as well as the pain, may be relieved by a mustard poultice or plaster over the epigastrium. After a considerable degree of improvement has taken place, a mineral acid with strychnine in a bitter infusion may be given thrice daily, an hour before meals.

ix. Perforating Ulcer of the Stomach

(Simple, Round, or Peptic Ulcer).

Etiology.—This disease is several times as common in women as in men. It is particularly common in female domestic servants, probably on account of their unsatisfactory mode of feeding. It is said that among men, those whose occupation involves constant pressure upon the epigastrium (*e.g.*, shoemakers and metal-turners) are specially liable. In females it is most common between

twenty and thirty years of age, and in males between thirty and fifty. It is often associated with chlorosis. The exact causation is still mysterious, but for some reason the tissue involved has its vitality reduced, and is thereupon digested by the gastric juice.

Morbid Anatomy.—Two varieties of ulcer are recognised : the *acute* and the *chronic ;* both involve the risks of hæmorrhage and perforation. The ulcer is almost as often multiple as single. Its most common seat is the posterior surface or lesser curvature near the pylorus. Not very rarely the duodenum is ulcerated as well as the stomach. The acute gastric ulcer is generally small and circular or oval, with soft edges, and well defined, as if punched out. The chronic ulcer generally measures $\frac{1}{2}$ to 1 inch across, but is sometimes much larger ; its edge is thickened and firm, and its shape varies. The floor of an ulcer may be constituted by the submucous, muscular or serous coat, or by a neighbouring viscus, such as the pancreas or liver. The artery supplying the ulcerated area has sometimes been found to be the seat of embolism or of endarteritis.

Ulcers very often heal completely, leaving small scars ; but a chronic ulcer may persist for years without healing. A large ulcer by cicatrising may cause serious obstruction if it is seated at the pylorus. In some cases an hour-glass constriction results from ulceration near the middle of the stomach.

If the ulcer extends as deeply as the serous coat, there is risk of perforation, but in most cases this has been anticipated by adhesions which form between the floor of the ulcer and neighbouring parts. If, however, the ulcer is on the anterior wall (which is unusual), adhesions do not form nearly so readily, and the risk of perforation into the peritoneum and consequent general peritonitis is great. Occasionally the ulcer perforates into the upper or lesser sac of the peritoneum, and gives rise to *subphrenic abscess* or *subphrenic pyopneumothorax*, or it may penetrate into the intestine.

Symptoms.—There may be no symptoms (*latent gastric ulcer*) ; or there may be slight discomfort after food with

occasional vomiting ; or there may be unmistakable symptoms, including pain, vomiting, and hæmatemesis.

Pain is very constant, and is often severe. Its maximum is usually about the middle line of the epigastrium, but it often extends through the body or round the left side to the back. It is generally associated with epigastric tenderness. It is often aggravated, either immediately or after a short time, by the taking of food, and the paroxysm then lasts till the food leaves the stomach, either by the pylorus or by being vomited.

Vomiting often follows the taking of food, and the vomited matter contains an excess of hydrochloric acid (*hyperchlorhydria*).

Hæmatemesis occurs in many cases, and is sometimes very alarming, but more frequently small in amount. The blood is red if abundant, but often it is scanty, and presents the appearance of coffee grounds, owing to the hæmoglobin being converted into hæmatin by the gastric juice. The stools may be tarry from the presence of altered blood (*melæna*). The bowels are constipated.

There is no pyrexia as a rule. Pallor is common, and the anæmia may be considerable, but there is rarely great emaciation. The tongue is clean. The urine often deposits phosphates.

Fatal hæmorrhage or perforation may set in with few or no preceding symptoms. The onset of perforation into the general cavity of the peritoneum is indicated by severe abdominal pain and shock. The patient lies on her back. The features are pinched, the pulse rapid and small, and the abdomen distended and tympanitic, even in the hepatic region. If the shock is survived, the symptoms of peritonitis develop.

Hour-glass Stomach was formerly regarded as due to a developmental defect, but Moynihan holds that it is always acquired, being the result either of chronic ulceration, or of malignant disease which is probably secondary to such ulceration. Moynihan[1] has put together the following signs, of which one or more may be present in cases of hour-

[1] *Brit. Med. Jour.*, 1905, i. 756.

glass constriction : (1) If the stomach is washed out with a measured quantity of water, the amount recovered will be less than the amount introduced, owing to a portion having passed into the pyloric pouch. (2) If the stomach is washed out till the water returns clear, a sudden rush of foul fluid may occur ; or if the stomach is washed clean, and the tube withdrawn and passed again a few minutes later, some foul matter may be obtained. These phenomena are due to regurgitation from the pyloric into the cardiac pouch. (3) There may be 'paradoxical dilatation'—*i.e.*, succussion sound may be present immediately after, as well as before, the apparent emptying of the organ by the tube. This is due to the fact that only the cardiac pouch is drained, so that the pyloric pouch remains capable of yielding the splashing sound. (4) Artificial distension of the stomach may cause at first a bulging in the left side of the epigastrium, but in the course of some seconds this gradually diminishes, while the right simultaneously becomes prominent. (5) If the two halves of a seidlitz powder are separately introduced into a normal or dilated stomach, no loud sound is heard by the stethoscope placed over the organ except at the pylorus ; but if there is an hour-glass constriction, a loud gushing sound can be heard 2 or 3 inches to the left of the middle line. (6) The area of gastric percussion is marked out, a seidlitz powder is given in two halves, and the stomach is again percussed. If there is an hour-glass constriction, there will be a great increase after twenty or thirty seconds in the cardiac pouch, while the pyloric pouch remains unaltered ; but in the course of a few minutes more there may be some increase in the pyloric pouch. (7) In some instances a distinct sulcus can be seen between the two pouches distended with carbon dioxide.

Diagnosis.—This is often easy, as when gastric pain and tenderness with hæmatemesis are complained of by an anæmic young woman. The hæmatemesis of chronic *alcoholism* is distinguished by the history, age, and facies of the patient. *Cancer* of the stomach rarely occurs in young women. Bleeding in cancer is usually in small

quantity ; hydrochloric acid is deficient ; emaciation and cachexia are the rule ; there is often tumour ; and the progress is different from that of simple ulcer. In *neuralgia* of the stomach pain is rather relieved than otherwise by the taking of food and by pressure on the epigastrium ; and there is no hæmatemesis.

Prognosis.—Many cases recover under treatment in a few weeks. Some of these will relapse after weeks, months, or years. Some patients continue to suffer for a long time from chronic ulcer. Dangerous hæmorrhage occurs in a small proportion of cases. Perforation is a still more formidable complication which cannot be foreseen, and is almost invariably fatal unless operation is resorted to. Not very uncommonly patients recover from symptoms suggestive of perforation, but it is probable that in such cases the ulcer has only reached the serous coat and caused a localised peritonitis. The cicatricial healing of an ulcer may cause serious trouble by obstructing the pylorus ; and adhesions to neighbouring parts may account for persistent pain. Chronic ulcer predisposes to cancer.

Treatment.—Absolute rest in bed for the first three or four weeks is essential, and for some weeks longer the patient should rest a great deal. At first the food should be entirely liquid. It should consist chiefly of milk diluted with lime-water or barley-water. Vegetable soup strained of all solids may also be given. The food should be given in small quantities every four hours. For two days after severe hæmorrhage, and also in the case of obstinate vomiting and pain after food, rectal feeding should be employed. When the symptoms have subsided, arrowroot, thoroughly mashed potato, and scraped beef may be added to the dietary ; and soon afterwards sweetbread, boiled white fish, and chicken may be given. Heavier articles of diet should be postponed for several months.

When pain is very severe, morphine may be given hypodermically, or bismuth and morphine may be given by the mouth. Vomiting may be relieved by half-drachm doses of bismuth internally, or by a mustard leaf or poultice over the stomach. The bowels should be regulated by warm

saline laxatives, and afterwards by cascara or aloes and iron. The pain in chronic ulcer may be relieved by nitrate of silver in pill, or by small doses of arsenic. For severe hæmorrhage the treatment is absolute rest, with morphine and ergotin hypodermically, and nothing but small quantities of ice by the mouth. In cases with a tendency to hæmorrhage, calcium should be regularly given. The saccharated solution of lime may be given in doses of ½ a drachm or more in milk, by the mouth or by the rectum ; or calcium chloride may be given in doses of 5 grains three times a day. It may occasionally be desirable to inject saline solution into the rectum, under the skin, or even into a vein. For perforation, prompt operation is necessary. Operation (especially gastro-enterostomy) may be justifiable if hæmorrhage recurs frequently, or if severe pain persists, in spite of prolonged and thorough treatment.

After the gastric symptoms have subsided, any existing anæmia must be treated. Secondary stricture of the pylorus may require operation.

x. Cancer of the Stomach.

Etiology.—With the doubtful exception of the uterus, no part of the body is so liable as the stomach to be the seat of carcinoma. Primary cancer is practically the only growth of importance in the stomach. About four-fifths of the cases occur between forty and seventy years of age. Males suffer more frequently than females. It has been supposed that heredity has some influence, as in the case of the first Napoleon, who, like his father, his brother Lucien, and two of his sisters (Pauline and Caroline), died of cancer of the stomach ; but with so common a disease, much may be explained by mere coincidence. Preceding chronic (simple) ulcer seems to predispose to cancer ; Mayo Robson states that in 59·3 per cent. of the cases of cancer of the stomach on which he has operated, the history pointed to antecedent ulcer.[1]

Morbid Anatomy.—In more than half the cases, the tumour is at the pyloric region, the next most frequent

[1] *Medical Press and Circular*, January 3, 1906, p. 9.

seat being the lesser curvature, and after that the cardia. Cancer of the stomach is met with under four types, viz., (1) *cylinder-celled epithelioma* (*adenocarcinoma*, *malignant adenoma*), a slowly growing tumour, usually involving a large part of the stomach before the patient dies, almost always ulcerating, and sometimes presenting a warty appearance at its margins; (2) *medullary*, *encephaloid*, or *soft cancer*, a soft tumour, very liable to ulcerate and bleed, frequently showing itself as a shaggy ulcer, and often giving rise to large secondary growths in the liver; (3) *scirrhus*, a hard growth which may ultimately involve a great part of the stomach, causing thickening of the wall and contraction of the organ, but not showing much tendency to ulcerate; (4) *colloid cancer*, sometimes involving more than half the wall of the stomach, which it transforms into a glancing material; not readily ulcerating; causing hardness but not contraction; and tending to spread both along the stomach and through the stomach to the peritoneum rather than to the lymph glands and liver.

Cancer of the pylorus often causes stenosis of that orifice, and thus leads to dilatation of the stomach. Cancer at the cardiac orifice may lead to shrinking of the stomach and dilatation of the gullet. Cancer of the stomach often leads to adhesions to neighbouring parts. Perforation is very uncommon. Secondary growths are very frequent, especially in the abdominal lymph glands and the liver.

Symptoms.—In many cases these are quite unmistakable, and point not only to the existence of malignant disease, but also to the stomach as the seat of the growth. Occasionally, however, there are no gastric symptoms, and the growth is only discovered after death. Or the case may be regarded as one of pernicious anæmia, though nowadays such a mistake is scarcely excusable. Sometimes only the secondary growths in the liver or elsewhere are recognised during life. A tumour is often felt after the disease has lasted for some months, but the diagnosis ought often to be made at an earlier stage than this.

The most important early symptoms are loss of flesh and strength, loss of appetite, and constipation, and if these

set in gradually in a person who has reached middle life, and has previously enjoyed perfect health, they almost certainly indicate cancer of the stomach, provided there is no distinct evidence of disease outside the digestive tract. It has been asserted that loss of the taste for smoking is a valuable early symptom, but this has not been my experience. Many of the localising phenomena (hæmatemesis, gastric dilatation, etc.) are due either to ulceration of, or to obstruction by the growth, and if both of these are absent, the direct evidence of gastric disease may be very scanty. Emaciation and weakness are often great, but considerable temporary improvement may take place under treatment, and the patient may appear to have regained almost complete health. There is often a lemon-yellow pallor of the skin as in pernicious anæmia, but the anæmia is generally of the chlorotic type. Slight pyrexia is common, but may cease under treatment. Epigastric pain and tenderness, anorexia, nausea, and a foul tongue are very common symptoms. The pain is usually aggravated by the taking of food.

Vomiting is generally present, occasional at first, but becoming more and more frequent as the disease progresses. Hæmatemesis is frequently observed, the blood being usually in small quantity and resembling coffee grounds. The taking of food may aggravate the pain, and may cause various other dyspeptic symptoms, such as flatulence, heartburn, etc. The vomited matter will contain food in various stages of digestion, often blood, sometimes sarcinæ if the stomach is dilated, but very seldom any free hydrochloric acid.

In a large proportion of cases a tumour can be felt in the epigastrium. Its situation naturally varies according to the part of the stomach involved. The stomach may be dilated. There may be enlargement of superficial lymph glands ; *e.g.*, above the clavicle.

Œdema of the feet is common as a result of the general debility, but sometimes marasmic thrombosis causes great swelling of one or more limbs. Ascites may result from obstruction of the portal vein, and jaundice from obstruction of the bile-ducts. Perforation of the cancer into the colon, with the formation of a gastro-colic fistula, may be indicated

by fæcal vomiting or by the passage of undigested food from the stomach through the rectum. The liver may be enlarged, and may present umbilicated nodules on its surface. The peritoneum may be thickened by the new growth. Secondary growths may develop in or beneath the skin—*e.g.*, at the umbilicus, as well as in superficial lymph glands. Death is usually due to exhaustion.

Diagnosis.—A tumour is often recognisable, and is in many cases conclusive evidence. It is important to bear in mind the combination of symptoms mentioned above, viz., the anorexia, constipation, and loss of flesh and strength setting in gradually, in a person who presents no signs of visceral disease, and at an age when cancer is probable. The absence of hydrochloric acid from the vomited matter is important, and so is the presence of lactic acid after a test meal. Hæmatemesis is very significant. In *gastritis*, the patient may have suffered from dyspepsia for a long time, and he may be able to recognise a distinct cause for his illness. There is no tumour or cachexia, and the case does not tend, as does cancer, to progress in an unfavourable direction in spite of treatment. In *simple ulcer*, hydrochloric acid is in excess, the tongue is clean, there is no cachexia and rarely tumour, bleeding is often profuse, and the age usually excludes cancer. Any question of *pernicious anæmia* ought to be settled by the examination of the blood.

Prognosis.—The ordinary duration is from six months to two years. There is no chance of recovery except through surgical intervention, and complete removal of the disease can scarcely be hoped for if a tumour can be felt through the abdominal wall.

Treatment.—The patient should take as much simple food as he can digest, and should avoid fatigue. If the stomach is dilated, it should be regularly washed out, and even apart from dilatation, this operation may give much relief. Bismuth and morphine may be given for severe pain and vomiting. Pylorectomy or gastrectomy is still a very formidable operation, and should not be attempted except in carefully selected cases. Gastro-enterostomy is theoretically a good operation for malignant stricture of the pylorus,

but it has fallen into some disrepute, as the immediate mortality is high, and the expectation of life in those who survive the operation is poor.

xi. Congenital Hypertrophy of the Pylorus.

This is a rare condition which has only recently been recognised. All the coats of the stomach at the pyloric portion of the organ may be involved, or the hypertrophy may be limited to the muscular elements. The obstructioh caused may be so great that even after death the gastric contents cannot be forced into the duodenum by compression of the stomach. The stomach and œsophagus are dilated.

Symptoms.—The child generally seems well for some days after birth, but after a period varying from days to weeks, vomiting sets in, and everything that is swallowed is rejected. Peristalsis of the stomach may be visible, and the thickened pylorus may be recognisable by palpation.

Treatment.—Medical treatment is usually unavailing, though recovery has followed a change from bottle-feeding to a wet-nurse. Surgical measures are frequently successful.

xii. Dilatation of the Stomach (Gastrectasis).

Acute Dilatation (*acute paralytic distension*) is rare, and occasionally fatal. It appears to result from the taking of great quantities of food and drink, and is perhaps favoured by existing acute or subacute gastric catarrh. The onset is sudden, and a leading symptom is the vomiting of an immense amount of liquid. The treatment consists in rest, emptying the stomach by the tube, and restricting the food and drink.

Chronic Dilatation is either (*a*) *obstructive*, or (*b*) *non-obstructive* or *atonic*.

Etiology.—(*a*) The first variety results from obstruction of the pyloric orifice by cicatrisation of a simple ulcer, cancer, congenital hypertrophy, kinking of the duodenum, etc. (*b*) Non-obstructive dilatation is seldom so great as the other variety. It is due to chronic inflammation of the

wall, habitual overloading of the organ, frequent gaseous distension, and loss of tone of the muscular wall in fevers and other debilitating conditions.

Symptoms.—These include thirst, pallor, weakness, emaciation and mental depression. Since but little fluid is absorbed from the stomach or passes on to the intestine, the urine is scanty, and the skin is dry. Constipation, uneasiness about the stomach, and various other dyspeptic phenomena are present. The mode of vomiting may be quite characteristic: once in several days the patient throws up a very large quantity of material. This is often frothy, and has the odour of stale beer. It contains *Sarcinæ ventriculi* (micro-organisms arranged in bundles like bales of wool tied with cord). Various symptoms may be due to the cause of the dilatation (*e.g.*, to cancer of the pylorus). Tetany is an occasional complication of gastric dilatation.

Physical Signs.—The enlarged stomach may be seen and felt in the abdomen, and the examination is facilitated if the organ is artificially distended with carbon dioxide. (See p. 476.) Visible peristalsis takes place spontaneously, or after friction of the abdominal wall. Percussion, auscultatory percussion, and succussion, as described in the section on examination of the stomach, give confirmatory evidence of dilatation. If the stomach contains sufficient liquid to yield dull percussion over part of its area, this area will change with the posture of the patient. Enlargement of the stomach must be distinguished from displacement (gastroptosis).

Prognosis.—Non-obstructive dilatation may be recovered from under appropriate treatment. When the pylorus is obstructed, the outlook is necessarily bad in cancer; and it is also bad in simple cicatricial cases if the obstruction is complete and not got rid of by surgical interference.

Treatment.—In *non-obstructive* dilatation, rest, regulation of the diet, treatment of catarrh, and the administration of strychnine are indicated. As a preliminary measure, the stomach should be washed out. In *obstructive* dilatation, the stomach should be thoroughly washed out, and for this purpose warm water faintly coloured with Condy's fluid may

be employed. This procedure should be repeated every day or two. Patients learn to do this themselves, and, indeed, may overdo it, on account of the relief it gives. Food should be given in small quantities and frequently; it should be mostly nitrogenous (starches, fats and liquids being restricted). Rectal feeding may be employed in addition. When the patient regains a fair state of health, gastro-enterostomy or some other surgical operation may be performed to give permanent relief.

xiii. Neuroses of the Stomach.

The functions of the stomach may be disturbed apart from any discoverable change of structure, and when such functional disturbance gives rise to symptoms, the condition may be described as *nervous dyspepsia*. Gastric neuroses may be motor, sensory, or secretory. The following are some of the most important disorders of this class:

Motor Neuroses.—The motility of the stomach may be either increased, as in (1) gastric cramp or griping (*tormina*); or diminished, as in (2) the atony which leads to non-obstructive dilatation. Overactivity is often caused by agents which irritate the stomach directly, such as excess of acid or undigested food. *Atony*, on the other hand, is commonly part of a general loss of tone which involves the nervous and muscular systems all over; it may therefore be induced by any agency which causes physical or mental exhaustion. It gives rise to flatulence and discomfort at the epigastrium during digestion. (3) *Hyperkinesis* or *supermotility* is an increase of the motor activity of the stomach which causes it to expel its contents into the duodenum too early. It causes no symptoms, and is recognisable only by the stomach-tube. (4) *Peristaltic unrest* is an exaggerated peristalsis, which sets in soon after the taking of food. The associated gurgling noises (*borborygmi*) audible to those around are distressing to the patient. (5) *Noisy eructations* (*eructatio nervosa*, *ærophagia*) may occur very frequently, the air having been previously swallowed. (6) *Nervous vomiting* occurs as a pure neurosis, without nausea. The *fitful*, *recurrent*, *cyclical* or *periodical vomiting* of children, a form of persistent

vomiting which occasionally ends fatally, is possibly of this nature, although it has been suggested that it is due to an auto-intoxication. It is sometimes relieved by frequent large doses of sodium bicarbonate (120-150 grains daily). Vomiting is a common symptom of intracranial disease. (7) *Merycism*, or *rumination*, is rare ; it occurs in idiots and some others. (8) The *gastric crises* of locomotor ataxy may be mentioned in this connection.

Sensory Neuroses.—The wall of the healthy stomach is devoid of sensation. It may be pinched or cut or cauterised without the patient knowing it, so that we cannot recognise any gastric neurosis characterised by diminution of sensibility. On the other hand, (1) *hyperæsthesia* is common. The mucosa may be too sensitive to mechanical and thermal impressions, so that the taking of food may immediately induce pain. Or, again, it may be abnormally sensitive to the hydrochloric acid of the gastric juice, so that the pain is felt when digestion is at its height, some hours after food. Or, again, the stomach may be the seat of the purely neuralgic condition (2) *gastralgia* or *gastrodynia*, which may be present even though the stomach is empty. Its paroxysmal character is sometimes simulated by the pains of simple ulcer or cancer. The gastric crises of tabes must of course be kept separate from simple neuralgia. Gastralgia occurs in neurotic subjects, especially females, and is sometimes periodic. It is often relieved by the taking of food and by pressure, and is seldom associated with vomiting. Gallstones, organic disease of the stomach and the gastric crises of tabes must be excluded by careful consideration of all the features of the case, but the diagnosis may be very difficult. (3) *Bulimia*, or excessive appetite, is sometimes a manifestation of hysteria and other diseases. (4) *Anorexia*, or want of appetite, also occurs in hysteria. An extreme form, which leads to serious consequences, is known as *anorexia nervosa*. (See under Hysteria.)

Secretory Neuroses.—Disturbances in the gastric secretion do not necessarily cause symptoms so long as they occur by themselves ; but if excess of hydrochloric acid happens to be accompanied by excess of sensibility, pain is induced ;

while if diminution of secretion is associated with diminution of motility, flatulence and discomfort are produced. (1) *Hyperchlorhydria* or *superacidity* is a condition in which the gastric juice secreted during digestion is abnormally rich in hydrochloric acid. It is apt to cause a sense of oppression or burning about the stomach about a couple of hours after food. There may be acid eructations, and sometimes vomiting which gives relief to the pain. The condition is common in neurotic and chlorotic subjects, and is a regular feature of gastric ulcer. (2) *Excessive secretion of gastric juice* may be either continuous or periodic. The former is the more common ; here the constant irritation of the stomach by the gastric juice, which is usually overacid, may lead to spasm of the pylorus, and thus to dilatation of the whole organ. Pain and acid eructations are among the symptoms. The periodic variety is rare ; it may be met with in neurasthenia. (3) *Subacidity of the gastric juice*, or even complete absence of free hydrochloric acid, may be met with as a neurosis in hysteria ; it is also met with in cancer and in atrophy of the mucosa. (4) *Achylia gastrica*, or complete absence of the gastric secretion, may occur as a neurosis, and also as a result of complete atrophy of the mucosa.

In the **treatment** of gastric neuroses, the general condition must be attended to, and any anæmia which is present must be treated by iron, with or without arsenic. In severe cases Weir Mitchell's treatment may be necessary. Bromide may be given along with iron and arsenic. Silver nitrate is another useful remedy for pain. Where excessive or highly acid secretion causes much suffering, the stomach should be washed out ; or large doses of alkalies may be given shortly before the pain is expected to become severe.

DISEASES OF THE INTESTINES.

i. Diarrhœa

(Looseness of the Bowels).

This symptom may be due to numerous causes. The stools have not the normal consistence, and are passed with abnormal frequency. The immediate cause is increase of peristaltic action, or increase of secretion, or both.

Diarrhœa may be due to purely nervous influences, as in students about to undergo an examination, or in those who are beginners in public speaking. Or the bowel may be stimulated by certain foods and drugs. Some drugs stimulate the glands, others stimulate the muscle, while concentrated saline solutions may drain fluid from the blood-vessels by osmosis. In amyloid degeneration, the vessels of the intestinal villi are unduly permeable, and diarrhœa is a result. Disease of the intestine, in the form of inflammation or ulceration, is a very common cause of diarrhœa. Looseness of the bowels is not uncommon when the large intestine is partly obstructed by cancer. In various morbid blood states, such as uræmia, the bowels may be loose. Diarrhœa with green stools is occasionally observed in pneumonia. In cholera, the stools resemble rice-water. The evacuations of enteric fever often resemble pea-soup, and similar stools are occasionally observed in other diseases. *Lienteric* diarrhœa is characterised by the passage of undigested food ; sometimes the patient has to rise from his meals to have an evacuation. The severe and exhausting diarrhœa of late phthisis is sometimes spoken of as *colliquative*.

Treatment.—The treatment of diarrhœa often consists in the removal of the cause—*e.g.*, of indigestible food by means of castor oil and opium, or of a catarrhal condition by appropriate diet and medicine. But diarrhœa should not be arrested without due consideration. For instance, in the dropsy of heart disease and kidney disease a certain amount of diarrhœa may be highly beneficial. If, however, it is desirable to check the symptom, the diet may be restricted to milk and milk foods ; while astringent remedies like lead

and opium, bismuth and morphine, dilute sulphuric acid, and chalk mixture with catechu may be administered. In some instances an enema consisting of 30 minims of laudanum in 2 ounces of mucilage of starch is very suitable.

ii. Constipation

(Costiveness).

In this condition, the fæces are retained in the bowel for an abnormally long period. This may result from deficiency of food, deficiency of intestinal secretion, or deficiency of action on the part of the intestinal muscle.

Etiology.—The causes may be local or general. Among the *local* causes are obstruction of the bowel by disease of its wall; atony of the bowel, which, it is to be noted, is itself a result of habitual constipation; pressure on the intestine, *e.g.*, by a tumour; and weakness of the abdominal muscles. The more remote or *general* causes include a family tendency; sedentary habits; errors of diet; lead poisoning; functional and structural diseases of the nervous system (hysteria, meningitis, etc.); diseases of the stomach; and various acute fevers. With many people, especially women, constipation is habitual, and this is largely due to want of regularity in attending to the calls of nature. Constipation is sometimes troublesome in little children without obvious reason.

Symptoms.—Some people with obstinate constipation feel none the worse for the retention, while others are quite miserable if they fail to obtain the regular movement. In exceptional cases, weeks or even months may elapse without any discharge. In habitual constipation, the fæcal matter gradually loses its watery portion by absorption, and tends to form hard masses or *scybala* which can often be felt in the colon through the abdominal wall. These accumulations may cause painful spasm, and also dilatation, inflammation, ulceration, and even perforation of the intestine. In the rectum they may cause discomfort; in the sigmoid flexure they may exert pressure on nerves, and cause pain in the

thigh. They favour the development of piles and of painful menstruation.

General symptoms, such as headache, mental depression, a furred tongue and foul breath may be present, but doubtless these phenomena and the constipation are often due to a common cause. It is of great importance to bear in mind the curious paradox that diarrhœa is often due to constipation. The irritation of the bowel accounts for the diarrhœa, and the hard fæces constitute a rigid tube through which the softer material passes.

Treatment.—Drugs should be avoided if possible. The patient should go to the closet at the same hour every day, preferably after a meal, even though there is no desire at the time, and even (in the case of young people) though some straining is needed for the first few days. The rectum can generally be educated to do its daily task in mankind just as it can in a dog or cat. The diet should not be concentrated, but should include vegetable matter which will confer bulk on the fæces, as well as fatty foods and liquid. An orange, apple or fig, or a glass of cold water, taken before breakfast, and oatmeal porridge at breakfast itself are valuable aids. Stewed fruits and salads at dinner-time are also to be recommended. Persons with sedentary occupations should take physical exercise when off duty.

If serious symptoms are present, it may be necessary at first to clear out the bowel by repeated doses of castor oil or olive oil, and repeated large enemata. Sometimes it is necessary to remove the hard fæces from the rectum by the finger, with or without the aid of some instrument, such as the handle of a spoon. The diet must then be seen to, and suitable medicines prescribed to aid it. A common practice is to take an aperient mineral water each morning some time before breakfast. The best remedy, however, is the liquid extract of cascara sagrada, of which the patient should take every evening enough to produce one evacuation on the following day. He may thus begin with 20 to 40 drops, and before long it may be found that the dose can be reduced very gradually until after some weeks the drug can be dispensed with altogether. The next best remedy, perhaps, is

aloes, which ought to be combined with iron in pill (extr. aloes, gr. i. ; ferri sulph., gr. ii. ; ft. pil.) ; one, or if need be two pills, to be taken thrice daily at first, and less frequently afterwards. Or the patient may take after dinner a pill containing extracts of nux vomica and belladonna, with aloin or extract of colocynth. In children, every endeavour should be made to secure a daily movement without drugs, by modifications of the diet, and by a regular resort to the water-closet. In an emergency with a little child, a small conical piece of soap may be inserted within the anus.

iii. Hæmorrhage from the Intestine.

Blood passed from the rectum may have its source in any part of the alimentary tract. The possibility of internal piles must be borne in mind. Blood in the stools is important evidence of ulcer of the stomach or duodenum, and when its source is so high up in the canal, its presence usually confers upon the fæces the colour and consistence of tar. The colour is due to sulphide of iron, and the condition is known as *melæna*. If, however, the blood is very abundant and passes rapidly through the bowel, it may be but little altered. Blood from the lower part of the small bowel or from the large intestine is not altered in this way. Sometimes in enteric fever, if the hæmorrhage is slight, the stools resemble strawberry jam in appearance ; if it is more abundant, the blood will be red or dark. Blood also appears in the stools in dysentery and in hæmorrhagic diseases such as purpura.

The causation of the dangerous *melæna neonatorum* is still obscure ; in one case examined by myself ulceration of the œsophagus was present.

Melæna must be distinguished from the dark condition of the stools which follows the administration of certain drugs, *e.g.*, iron and bismuth.

The treatment of intestinal hæmorrhage must have reference to its probable cause.

iv. Intestinal Colic.

Etiology.—Intestinal colic is a painful spasm of the bowel, and is generally due to irritation by something which cannot be digested by the particular individual. It is one of the characteristic features of lead poisoning (*lead colic*, *painter's colic*). Purgatives often cause it (*tormina*, *griping*). Colicky pains may result from spasm of the intestine above any obstruction (*e.g.*, cancer, fæcal accumulation, etc.).

Symptoms.—Pain is the principal symptom, and may be severe. The bowels may be bound or loose.

Diagnosis.—This must be very certain before any active treatment is employed. Lead poisoning is recognised by the blue line on the gums, but it is of special importance to exclude intestinal obstruction and inflammatory processes about the abdomen. The absence of fever and of shock, and a history of having eaten some indigestible article point to colic.

Treatment.—If the pain is certainly due to indigestible matter, a dose of castor oil with laudanum should be given. If there is doubt, purgatives must be avoided. An india-rubber foot-warmer should be placed over the abdomen, and an enema may be administered. A dose of morphine may be given (unless the patient is a young child) to relax the spasm ; it may thus prove to be the best laxative.

v. Catarrhal Enteritis.

Etiology.—Inflammation of the mucous membrane of the bowel is excited by unsuitable articles of diet, such as tainted milk, unripe and overripe fruit, etc. ; by violent purgatives ; and by irritant poisons. It is sometimes produced by chill, but it is specially frequent in hot weather—probably because at such seasons food becomes readily tainted, and fruit is eaten in bad condition. Intestinal catarrh results also from the passive hyperæmia of heart disease and hepatic cirrhosis. It is sometimes met with in pneumonia and other infections.

Morbid Anatomy.—The usual changes are redness and swelling of the mucous membrane, with increase of secretion. There is cellular infiltration of the mucosa. There

is swelling of the solitary and agminated follicles, and sometimes these become eroded so that follicular ulcers develop. The inflammation usually passes off, but it may become chronic, and lead to thickening or thinning of the mucosa.

Symptoms.—Diarrhœa is the principal or, it may be, the only symptom. It is often associated with griping pains due to spasm of the bowel, and with *borborygmi* or rumbling noises. The stools vary greatly in consistence and colour ; they may be pale and watery. The watery character is attributable, partly to the increased amount of intestinal secretion, and partly to the intestinal contents being hurried on to the rectum by the increased peristalsis before the liquid portion can be absorbed. Anorexia and thirst are common, and there may be considerable depression, but there is little or no fever. Acute attacks usually pass off in a few days.

Chronic symptoms of milder type may follow an acute attack, or may develop gradually as a result of passive hyperæmia.

When the *small intestine* is chiefly affected, the stools will contain undigested food (*lienteric diarrhœa*) ; if the *colon* is at fault, the stools will be slimy from the presence of mucus ; when the *rectum* is inflamed, *tenesmus* (constant desire to go to stool) will be a symptom.

Treatment.—If the attack has just begun, and is clearly traceable to some improper article of diet, a purge of castor oil and laudanum should be given ; but often this is not required. Rest in bed, warmth, and milk diet are the essentials of treatment. Chlorodyne or tincture of opium (5 to 10 drops for an adult) may be given every four hours till the diarrhœa is checked, but infants of less than one year should be treated without opiates as a rule. Bismuth is another useful remedy.

In *chronic* cases, bismuth, Dover's powder and lead and opium pill are suitable remedies. In obstinate cases, warm water enemata, containing silver nitrate, lead acetate, or some other astringent, should have a trial.

vi. Enteritis of Children (Dyspeptic Diarrhœa).

Etiology.—It is customary to distinguish enteritis as it occurs in young children from that which is met with in older children and adults. In infants the most important cause of this disease is improper feeding, and accordingly it is chiefly met with in hand-fed children. In babies, it may set in soon after birth. It may occur in the course of the first two or three years in children who are fed on 'the run of the house,' viz., on the bacon, potatoes, bread and tea which constitute the dietary of their parents. Tainted milk is naturally a common cause. Starchy foods cannot be digested by young babies, and when taken by them, are apt to decompose, and irritate the digestive tract. Chill, and the general disturbance associated with teething and rickets, favour the disease. Next to improper feeding, the most important factor in etiology is hot weather.

Although many kinds of organisms are found in the stools in infantile diarrhœa, no one of them seems to be specific.

Morbid Anatomy.—The principal change is enlargement of the lymphatic follicles, which is sometimes associated with ulceration.

Symptoms.—Among these are diarrhœa, griping pains, and sometimes vomiting. The stools appear like chopped green vegetables, and may be slimy with mucus. In severe cases, death may occur in a few days. Or the case may become subacute or chronic, and emaciation takes place. Improvement may set in after several weeks, or death may result from collapse, asthenia, or broncho-pneumonia.

Treatment.—If the child is on the breast, some of the milk should be drawn off by the breast-pump and examined to ascertain whether it is of good quality; and if it is not satisfactory, a wet-nurse should be obtained, or the child must be fed artificially. Even a young baby should not be fed oftener than once in two or two and a half hours. If woman's milk is not available, the usual substitute is cow's milk diluted and sweetened. The milk should be sterilised

by heat as soon as it is brought into the house, the best way being simply to bring it to the boiling-point. When about to be used, it is diluted by the addition of water, and to this some lime-water should be added so as to diminish the size of the curds formed in the stomach. For the first two or three months, one part of milk may be diluted with two of water; in the next few months, equal parts may be used; and thereafter, until the time of weaning, two parts of milk may be diluted with one of water. Very robust infants, however, will thrive on milk which is less diluted than that which has just been recommended. The quantity of milk required will vary according to the appetite of the child, but each meal should be specially prepared, and whenever it has been taken, the bottle, teat, etc., must be thoroughly cleansed with warm water.

If the diarrhœa persists, in spite of a carefully regulated milk diet, it is sometimes well to dispense with the milk altogether for two or three days, and resort to barley-water or white of egg and water. Small doses of grey powder (1 grain for a baby) with sodium bicarbonate and sugar of milk may be given once or twice a day as an intestinal disinfectant. In severe diarrhœa, it is sometimes right to give laudanum, (1 minim for a child of twelve months; ½ minim for an infant of six months) twice or thrice daily. For a state of collapse or great asthenia, old brandy may be tried.

vii. Cholera Nostras

(British Cholera. Sporadic Cholera. Choleraic Diarrhœa).

Etiology and Morbid Anatomy.—This is an acute affection which is met with in hot weather in temperate climates. In some cases, indigestible or decomposing food appears to be the cause, but in others the causation is obscure. The post-mortem changes are trifling, consisting chiefly of pallor and emptiness of the stomach and bowels, and occasionally minute hæmorrhages. There is therefore little evidence of inflammation, and it is supposed that the symptoms are due to a bacterial poison, either generated in the alimentary canal or taken into it by the mouth.

It was formerly supposed that a spirillum described by Finkler and Prior might be the cause, but this is now doubted.

Symptoms.—These set in suddenly with vomiting and diarrhœa. The stools are large and soon come to resemble rice-water. Thirst, cramps in the calves, coldness of the surface, sunken eyes, and scantiness of urine are other symptoms. After a day or two, death may take place, but a speedy recovery is the rule.

Diagnosis.—The clinical difference between this disease and Asiatic cholera is one of degree, and the distinction is sometimes purely bacteriological. (See under Cholera.)

Treatment.—It may be impossible at first to give anything by the mouth or rectum. Morphine may then be given hypodermically to relieve pain and allay the irritability of the digestive tract. Fomentations should be applied over the stomach. If vomiting is not severe, iced champagne, iced brandy, Dover's powder or laudanum may be given by the mouth, and the patient may possibly retain small quantities of iced milk or barley-water. For severe collapse, several pints of physiological saline solution should be injected under the skin or even into the veins.

viii. Cholera Infantum.

Cholera infantum is probably the same disease as that just described. It attacks children in hot weather, especially in large towns. Vomiting, diarrhœa with rice-water stools, thirst and collapse are leading symptoms. The temperature in the rectum may be several degrees higher than that in the axilla. The great loss of fluid from the body and the resulting cerebral anæmia may lead to the condition termed *hydrocephaloid*. This is characterised by depression of the anterior fontanelle, stupor, changes in the pupils, and sometimes fatal coma. This form of diarrhœa in children is very fatal.

The treatment is on similar lines to that recommended for cholera nostras.

ix. Membranous Enteritis

(Croupous or Diphtheritic Enteritis).

This disease is characterised by necrosis of the mucous membrane, either quite superficially or to any depth. It may be confined to the most prominent parts of the folds. The condition is met with in acute infections such as pneumonia, in the last stages of chronic diseases, *e.g.*, Bright's disease, heart disease, etc., and as a result of irritant poisoning, *e.g.*, by arsenic or mercury.

In cases of poisoning, the symptoms may be those of catarrhal enteritis ; in the other groups of cases, they are apt to be masked by the phenomena of the primary disease.

x. Phlegmonous Enteritis.

Phlegmonous enteritis is usually a localised disease, resulting from intussusception, strangulated hernia or some neighbouring inflammation. All the coats of the bowel are involved in the inflammation, the serous coat being hyperæmic and perhaps covered with lymph. The disease may, however, occur without obvious cause and lead to suppuration in the submucous tissue.

The symptoms are commonly those of peritonitis, and unless hernia or intussusception coexist, a correct diagnosis can scarcely be made.

xi. Simple Ulcerative Enteritis and Colitis.

Various forms of intestinal ulceration have already been described, such as enteric, tubercular, dysenteric, follicular (in catarrhal enteritis) and fæcal (in constipation). The peptic ulcer of the duodenum and the cancerous ulcer will be alluded to hereafter.

Apart from all these, the small intestine occasionally and the large bowel more frequently are involved in extensive ulceration of an apparently simple kind. Large areas of mucosa and submucosa may be destroyed, and perforation may ensue. The disease attacks adults.

Symptoms.—Diarrhœa is the chief symptom, and there is frequently pain in the abdomen, but seldom tenesmus. The

stools sometimes contain blood, but there is little or no mucus. The disease occasionally becomes chronic, but more often it proves fatal in the course of a month or two, from exhaustion or from perforation.

Treatment.—The treatment is similar to that recommended for chronic dysentery, and will include a carefully arranged dietary, bismuth, lead and opium by the mouth, and warm injections by the rectum.

xii. Mucous Colitis

(Membranous Enteritis. Mucous Colic. Tubular Diarrhœa. Myxoneurosis Intestinalis).

The distinguishing feature of this curious disease is the discharge from the rectum of membranous-looking casts of the bowel. The membrane consists not of fibrin but of tough mucus, which may be in shreds, or may form a complete tube extending to many inches in length. The disease is a secretory neurosis of the colon, whose mucous membrane presents no change. It is much more common in women than in men. The patients are often neurotic subjects, but in the case of women there may be pelvic disease. Constipation is common. The casts are passed from time to time during years. As the membrane adheres closely to the mucosa, it can easily be understood that if a small piece becomes loosened, it will excite the action of the bowel, so that there will be a constant dragging upon, and irritation of the wall of the gut, until the loosened portion of the cast breaks away altogether. Such an occurrence gives rise to recurring paroxysms, which may be associated with severe abdominal pain and even with localised tenderness suggestive of appendicitis ; but the absence of fever, the progress of the attack, and the characteristic evacuations make the nature of the seizure clear. There may, however, be no symptoms referable to the passage of mucous casts from the bowel.

Treatment.—The key to the successful treatment of this disease is to give a diet which will cure the constipation, namely, a diet which leaves a bulky, indigestible residue. This bulky mass appears to act by gently scraping the

mucosa as it passes along the bowel, and thus preventing the mucus from consolidating into tough tubes. For this purpose the diet should consist largely of fruits, vegetables, oatmeal porridge, bread, nuts, etc. The patient, indeed, would do well to be a vegetarian every second day, or even for a time continuously. Treatment on these lines yields very good results. If, however, a case proves obstinate, an opening may be made into the ascending colon, or, perhaps better, into the appendix. This is kept open for some time, so as to give the large bowel rest and allow of its being regularly flushed out from above downwards.

xiii. Sprue

(Psilosis).

Sprue is a chronic disease of warm climates, practically confined to adult Europeans, and characterised by flatulent dyspepsia, profuse liquid or clayey stools, and usually a red, eroded condition of the mucous membranes of the mouth and gullet.

Morbid Anatomy.—After death there is found atrophy and superficial erosion of the mucosa of the digestive tract, from mouth to anus, either in patches or universally. There may also be fibrosis of the submucosa.

Symptoms.—The first symptom is usually diarrhœa. After a time the stools increase in number, bulk and pallor. The mucous membrane of the mouth becomes eroded and exceedingly sensitive ; salivation is present, and for a similar reason, swallowing may be painful. Emaciation and weakness set in, but temporary improvements take place from time to time. At last, however, after years, the patient, unless proper treatment has been carried out, dies from exhaustion, severe diarrhœa or intercurrent disease. In some mild cases, the general health is not much impaired.

Treatment.—The treatment consists in an exclusively milk diet for many weeks to begin with, the milk being sipped in small quantities. After a time the dietary may be cautiously extended. If milk fails, meat juice may be tried for a time. Medicines are of little use. The patient should of course keep away from tropical climates.

xiv. Perforating Ulcer of the Duodenum.

The peptic ulcer occurs not only in the stomach, but also in the lower part of the œsophagus and first part of the duodenum. It occurs, therefore, in those parts to which the acid gastric juice has access. It is occasionally met with in the jejunum as a consequence of gastro-enterostomy. Ulcer of the duodenum is much less common than gastric ulcer It is far more frequent in males than in females. It occasionally follows superficial burns.

Symptoms.—The clinical features are in the main identical with those of gastric ulcer as already described, and the two are not very rarely present at the same time. According to Moynihan, the characteristic pain of duodenal ulcer is the 'hunger pain' which sets in from two to four hours after the completion of a meal. If another pain comes for a time at an earlier stage of digestion, it suggests that a gastric ulcer is present in addition. Other facts which might suggest the diagnosis of duodenal rather than of gastric ulcer are these: pain seated in the right hypochondrium; tenderness at the right side of the back below the ribs; and repeated attacks of melæna without hæmatemesis.

Diagnosis.—The pain of gallstones may set in some hours after food, but it is not likely to do so after every meal, and, moreover, as Brunton suggests, neutralisation of the acid gastric juice by the administration of an alkali before the chyme begins to pass into the duodenum may greatly diminish the pain of duodenal ulcer, whilst it is not likely to modify the pain of gallstones.

Treatment.—The treatment is similar to that of gastric ulcer.

xv. Appendicitis.

(EPITYPHLITIS).

Definition.—A form of peritonitis, local or general, starting from the vermiform appendix, and corresponding clinically to the conditions formerly known as *typhlitis*, *perityphlitis*, *paratyphlitis*, and *iliac phlegmon*.

Etiology.—Microbes are the immediate cause, and the most common appears to be the *Bacillus coli communis*, which is

normally present in the intestine, but owing to the predisposing causes of the disease becomes pathogenic. Other organisms may be associated with it, including the *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes* and *Pneumococcus*.

Among the causes of appendicitis are catarrh resulting from imperfect emptying of the cæcum and appendix, a condition which may be due to constipation or to overeating. Fæcal masses, and occasionally foreign bodies, such as the seeds of fruits, may irritate the appendix. The inflammation may be due to extension from the right ovary and Fallopian tube, or it may result from tubercular ulceration. Cases (described as *rheumatic*) sometimes follow a chill. It is supposed that the most important factor in the etiology is the blocking of the lumen of the appendix, with the result that the virulence of the enclosed microbes becomes increased.

Most cases occur in adolescents and young adults. Males suffer more than females.

Morbid Anatomy.—This varies much. In simple cases there is catarrh of the mucosa, with excess of mucus, which, together with microbes, fills up the lumen. Slight ulcerations may now occur, and thus permit the microbes to reach the submucous layer, and here again the process may cease. But an abscess may form in the wall and cause a local peritonitis, or the ulcer may perforate into the peritoneal cavity. In yet other cases, a portion or the whole of the appendix becomes gangrenous.

In the simpler cases of appendicitis, repeated attacks of inflammation may obliterate the lumen of the appendix, and the appendix itself may become buried in a mass of fibrous adhesions.

The usual result of perforation is the formation of an appendicular abscess, which, being primarily intraperitoneal, is a localised suppurative peritonitis. This abscess may burst through the anterior abdominal wall, or into the cæcum, or into the general peritoneal cavity or elsewhere. The contents have usually a fæcal odour.

Acute general peritonitis results if perforation or necrosis takes place in the absence of adhesions. It may also be due to extension from, or bursting of, an appendicular abscess.

In a considerable minority of cases of appendicitis, fæcal concretions (*coproliths*) are present in the appendix. These have usually originated within the appendix itself, and it is probable that in some instances they are the actual cause of the disease. Some of these concretions are very like fruit-stones in appearance. Foreign bodies, such as pins, may also be present in the appendix in this disease.

Symptoms.—In many cases the mode of onset is characteristic, and this is specially true of the simpler cases. Pain suddenly sets in over the abdomen generally, in a young person, and in the course of some hours becomes restricted to, or most severe in, the right iliac region. This region is also tender, particularly at McBurney's point, which corresponds to the outer edge of the right rectus muscle in a line drawn from the umbilicus to the anterior superior iliac spine. In many cases, some induration can be felt, but the recognition of this may be difficult owing to local tenderness and muscular rigidity. There is moderate fever with acceleration of the pulse, constipation, sometimes vomiting at the outset, and occasionally trouble with micturition.

Several varieties of cases may be distinguished :

(1) *Appendicular colic* is an expression which has been applied to cases characterised by attacks of pain and tenderness, with their maximum intensity at McBurney's point, but without physical signs, and, it may be, without fever. Some have supposed that these attacks are due to spasm of the muscle of the appendix, excited by partial occlusion, but others hold that they are associated with the early structural changes of appendicitis.

(2) *Mild cases with the typical symptoms already described* are very common. There is slight swelling, or none at all, and recovery takes place after a few days.

(3) *Appendicitis with abscess formation* is characterised in the early stages by the usual symptoms, but later on, in spite of rest, the local tenderness continues, the tumour increases, and hectic fever persists. There may or may not be rigor, with local redness, œdema, and fluctuation. A well-marked leucocytosis which steadily increases is very suggestive of pus.

(4) *Acute perforative, fulminating*, or *gangrenous appendicitis* begins with acute abdominal pain. There is great tenderness

in the right iliac region. In many cases, repeated vomiting is a feature. The signs of collapse quickly appear, completing the picture of *peritonism*, and death ensues within forty-eight hours. The patients have been in good or fair health until these symptoms suddenly develop, and life may be prolonged for but a few hours after their commencement.

(5) *Relapsing appendicitis* is very common. After being quite free from symptoms, and it may be from local tenderness and induration as well, for weeks, months or years, the patient is seized with another attack similar to the first. The frequency of the recurrences may render the patient a chronic invalid. After several attacks there may be final recovery, or perforation may take place, or an abscess may form and rupture into the abdomen.

Complications.—Among these may be mentioned pylephlebitis, hæmorrhage from perforation of a large vessel, septicæmia and parotitis.

Diagnosis.—The mode of onset, as already stated, is often sufficiently characteristic. There may, however, be difficulty in distinguishing the disease from some forms of *intestinal obstruction*, but in the latter the temperature is low, and there may be fæcal vomiting and bloody stools ; whereas in appendicitis, the temperature is generally elevated, fæcal vomiting and bloody stools are not observed, and the symptoms point to peritonitis rather than to obstruction. In a case of *mucous colitis* the symptoms may for a time be strongly suggestive of appendicitis, but there is no fever, and mucous casts ultimately appear in the stools. In one case where the diagnosis of mucous colitis was clear, the patient had already had her appendix needlessly removed, of course without cure. In *enteric fever*, the mode of onset is usually quite different from that of appendicitis ; moreover, the course of the temperature, together with Widal's reaction, should suffice for the differential diagnosis. If the diagnosis is not perfectly clear, a rectal examination ought to be made.

Prognosis.—The great majority of cases end in recovery. The simple cases (group 2 above) almost invariably recover. Of cases with abscess, it is estimated that about a third die. The cases in group 4 generally prove fatal. Many cases which recover suffer from one or more relapses,

and a relapse seems to be nearly as dangerous as a first attack. A relapse may be due to errors of diet or to muscular exercise, but it may occur in spite of the greatest care. It should be borne in mind that the mildest case may become severe, and that at the onset of symptoms, such as occur in simple cases, the peritoneum may be already extensively involved.

Treatment.—For the simple cases, absolute rest in bed, a diet restricted to diluted milk, and a soap-and-water enema every second day may be all that is necessary. If the pain is severe, leeching, belladonna and glycerin, and hot fomentations are suitable local remedies. No more opium should be given than is required to relieve severe pain and permit of sleep. If under favourable conditions as to rest, diet, and nursing, the pulse and the local tenderness do not, within a period of from two to four days, present a decided change for the better, it may be concluded that suppuration has taken place, and that operation is essential. When this is recognised, there should be no delay in operating. In acute perforative and gangrenous cases, the only hope of recovery depends upon the promptness with which an abdominal section is performed. If relapses are so frequent or formidable as to keep the patient in constant ill-health or entail much suffering, the diseased appendix should be removed at a time when there are no acute symptoms; the danger of the operation is less than that which would accompany another relapse.

xvi. Intestinal Obstruction.

Etiology.—The causes of intestinal obstruction may be classified as follows: (1) impaction of contents; (2) intussusception; (3) volvulus; (4) stricture; (5) strangulation by bands or in apertures; (6) compression and traction.

(1) The *intestinal contents* which cause obstruction are chiefly gallstones and fæces. Obstruction by gallstones occurs chiefly in the second half of life, and is more common in women than in men. The usual situation is the lower part of the ileum. The large gallstone reaches the bowel by ulcerating through the gall-bladder.

Obstruction by fæces may occur at any age. The accumulation is in the large bowel, and women are most liable.

Much more rarely foreign bodies, such as fruit-stones, artificial teeth, accumulations of bismuth or magnesia, and hairs which the patient has swallowed give rise to obstruction. Intestinal calculi (*enteroliths*), such as fæcal concretions (*coproliths*) and stones formed around grains of oats (*avenoliths*), may also be mentioned as rare causes.

(2) *Intussusception* or *invagination* arises through a portion of bowel slipping into the part immediately lower down. In the intussusception there are three layers: the inner or *entering* layer, the middle or *returning* layer, and the outer or *receiving* layer. The cylindrical tumour thus produced varies in length from less than an inch to more than a foot. It grows in size by the middle and inner layers, which keep pace with one another, passing further into the lumen of the bowel and dragging after them the upper end of the outer layer.

The part involved may be either small intestine (*enteric*), or large intestine (*colic*, *rectal*). The most common form is the *ileo-cæcal*, in which the ileo-cæcal valve enters the colon. In the *ileo-colic* form, the lower part of the ileum passes through the valve.

The bloodvessels going to the inner and middle layers of the intussusception are compressed, so that congestion and œdema result, with hæmorrhage from the mucous surface. If reduction is not effected, gangrene of the invaginated bowel may take place, and if adhesions have previously formed all round between the middle and inner layers, a cure may result.

Intussusception is most common in childhood, and is more frequent in males than in females. It may arise in connection with diarrhœa or constipation, but there is often no obvious cause. The immediate agency in its production is irregular peristalsis.

(3) *Volvulus* or *twist* is most common at the sigmoid flexure. The ends of the loop of bowel are fixed, and the loop becomes twisted so that its one limb crosses over and compresses the other. Sometimes a piece of bowel is twisted on its own axis. Or the cæcum may be bent upwards. Or again, one loop of bowel is twisted round another loop.

Volvulus is most common in middle life, and affects males more often than females.

(4) *Stricture* is chiefly concerned in chronic obstructions, though it sometimes causes acute symptoms. Cicatricial stenosis is usually in the large intestine, and may follow tubercular, syphilitic, dysenteric, and catarrhal, but very rarely enteric ulceration. Occasionally it follows an injury.

New growths, whilst occasionally simple, are usually malignant. They are generally cylindrical epitheliomata which give rise to a dense ring around a short length of bowel. The lumen may be so narrowed as not to pass the tip of the little finger. Cancer is met with chiefly in the large intestine, and is specially common about the flexures. The patient has generally reached middle age.

Above the stricture, whether simple or malignant, the bowel becomes hypertrophied and dilated. Ulceration and perforation may eventually take place.

(5) *Strangulation* is the most common cause of acute obstruction. A loop of bowel may slip under a band of adhesions, which are usually the result of old peritonitis, either in the pelvis or elsewhere ; or under a Meckel's diverticulum which has become adherent at its end to the abdominal wall or the mesentery ; or under an adherent appendix ; or under the pedicle of a tumour ; or through a slit in the omentum or mesentery. It is almost always the small intestine which is strangled.

Most of the cases are males, and a large proportion occur in the first half of adult life, though Meckel's diverticulum may cause trouble at an earlier age. In this and every other form of acute obstruction, the bowel is greatly distended above the obstruction and collapsed below it.

(6) *Compression* and *traction* are very uncommon causes. Compression may be due to a tumour of a neighbouring organ, or to a neighbouring coil of bowel filled with hard fæces, or to old peritonitic adhesions. Traction by adhesions, or by the enlargement of a neighbouring tumour, may cause kinking of the large or small bowel.

Symptoms of Acute Obstruction (*e.g.*, from strangulation, intussusception, volvulus, or an impacted gallstone).—The most important are pain in the abdomen, constipation and vomiting. The pain sets in abruptly and often without apparent cause. It is generally in the umbilical region,

whatever the seat of the obstruction. It is often intermittent until the obstruction is complete; and thereafter it is continuous though subject to exacerbations. There is usually no tenderness until peritonitis sets in. The pain is soon followed by vomiting with characteristic features, viz., first of stomach contents, then of bilious matter, and finally of liquid with a fæcal odour. The constipation may be absolute from the first, so that not even flatus is passed. This is partly a reflex phenomenon. If the strangulation is not very severe, fæces may be passed for a time, and in intussusception, these may be mixed with blood and mucus. If the obstruction is not high up, the abdomen frequently becomes distended. The general phenomena of collapse are present, including a pinched look, a cold, moist skin, a quick, small pulse, a subnormal temperature, a dry tongue, thirst, and scantiness of urine. The case generally ends fatally in from three to six days, from exhaustion or from peritonitis.

Symptoms of Chronic Obstruction (*e.g.*, from fæcal impaction, simple or malignant stricture, traction, or pressure by a tumour outside the bowel).—The symptoms develop slowly. The pain is at first slight and comes at long intervals ; it may be attributed to dyspepsia. Constipation is often the most troublesome symptom, and the bowels may never act properly without medicine. Occasionally, however, there is a daily loose motion, or diarrhœa alternates with constipation. The formed stools may be narrow and suggestive of a diminished calibre of the bowel. There may be pain, tenderness and increase of resistance over the seat of obstruction ; and distension and peristalsis may be recognisable in the bowel above. Acute symptoms may threaten from time to time and pass off, it may be for years ; and even after complete blocking has taken place, vomiting may be long postponed. In cases of malignant stricture the general health is often impaired, but sometimes this can scarcely be recognised even by the patient's friends. When complete obstruction has supervened in chronic cases, death may take place speedily as in acute obstruction, or may occur after a week or two from exhaustion or from perforation.

It is to be noted that malignant disease of the bowel may be associated with attacks of fever, which are doubtless due

to absorption, and also with free hæmorrhage. Thus more than one case has come under my notice where cancer of the splenic flexure in men, aged between fifty and sixty, was associated with inability to lie comfortably on one side owing to pain or uneasiness induced thereby in the left hypochondrium; with irregular attacks of fever; and with one or more considerable hæmorrhages from the intestine. The

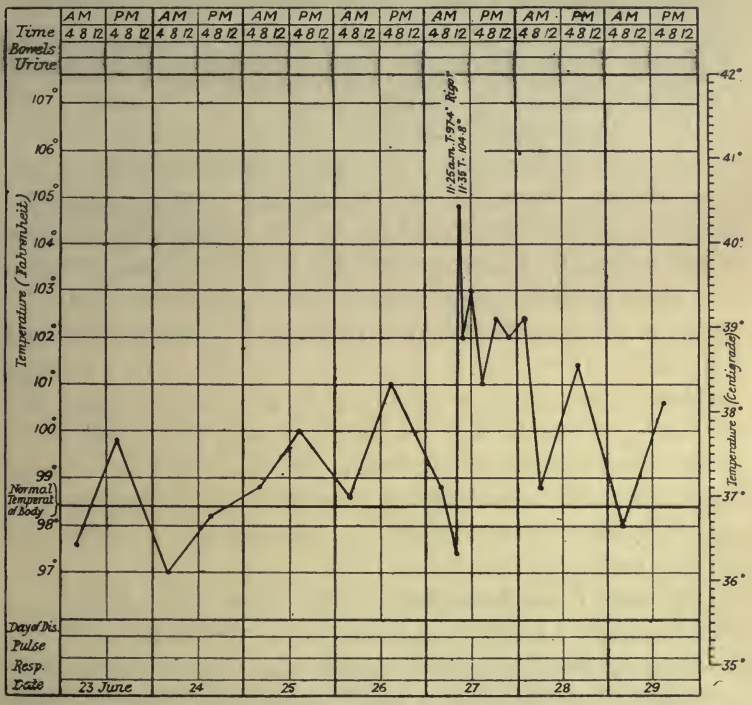

FIG. 32.—FEBRILE PAROXYSM IN CANCER OF THE SPLENIC FLEXURE OF THE COLON.

accompanying temperature chart shows one of these febrile paroxysms (Fig. 32).

Diagnosis of the Seat of Obstruction.—Rectal examination may disclose an intussusception, a malignant stricture, or a mass of hardened fæces. Local tenderness, induration, and muscular rigidity may indicate the seat of a malignant growth. Distension of coils of bowel and visible peristalsis may show how far down the permeability of the bowel extends. Obstruction of the colon low down displays the

horse-shoe arrangement of the large bowel above the lesion. Obstruction of the small bowel, or at the cæcum, causes distension of loops of small bowel, especially in the lower central part of the abdomen. Tenesmus and the passage of blood and mucus suggest obstruction of the large bowel; whereas the early onset of fæcal vomiting suggests that the lesion involves the small intestine.

Diagnosis of the Cause of Obstruction.—This may be impossible, but must be attempted. The acute or chronic character of the symptoms is the first point in diagnosis. Further, a history of peritonitis, or of tuberculosis, or of disease in the female pelvis would raise the question of bands, compression or traction. An injury might have led to rupture of the omentum, or to bruising and cicatrisation. Intussusception occurs chiefly in childhood, strangulation occurs especially in early adult life; gallstone impaction is met with mostly after middle life. Intussusception can often be clearly recognised. There is a cylindrical tumour, generally in the region of the transverse colon; and tenesmus and bloody mucous stools may also be present. Occasionally the tumour can be felt in the rectum. The condition is sometimes subacute or chronic. In fæcal obstruction, the hard fæces can be felt in the colon and in the rectum. In gallstone obstruction, there is occasionally a history of hepatic colic.

Diagnosis from Appendicitis.—In appendicitis there may be a history of a previous attack; the abdominal pain and tenderness tend to settle in the right iliac region; the temperature is elevated; vomiting takes place, as a rule, only at the commencement and is not fæcal.

Treatment.—Purgatives must be avoided. Morphine and atropine may be given hypodermically to relieve severe pain, but opiates must be used in the smallest doses that will make the pain tolerable. Hot fomentations or ice poultices may be applied to the abdomen. For severe vomiting, the stomach should be washed out several times a day. In acute obstruction, the patient should be fed as much as possible by nutrient enemata, nothing being given by the mouth except small pieces of ice. In intussusception and fæcal impaction, large enemata are indicated, and these may also

be useful if fæces have accumulated above a stricture. But in all cases of acute intestinal obstruction, the advice of the surgeon should be sought as quickly as possible, for in many instances operation offers practically the only hope, though, perhaps, not a very brilliant one, of saving the patient's life. Moreover, if an operation is required, delay adds to the danger.

In chronic cases, in addition to careful dieting, exercise, and regulation of the bowels, surgical measures may occasionally be of service.

xvii. Enteroptosis

(Splanchnoptosis. Glénard's Disease).

Displacement of individual viscera of the abdomen—for instance, of a kidney—through relaxation of their supporting structures, is quite common. Much less frequently there is drooping or falling of the abdominal viscera in general, and this it is which is known as Glénard's disease, or enteroptosis.

Etiology.—Glénard's disease is most common in females, and especially in young and neurasthenic women. Some of the cases are explained by relaxation of the abdominal walls in consequence of numerous pregnancies. The displaced stomach is well seen after distension with carbon dioxide. Even in well-marked cases of this kind, there may be no symptoms. In another group of cases, there is elongation of the bands of peritoneum, bloodvessels, and other structures by which the organs are suspended. This is doubtless often due to tight-lacing or to the wearing of heavy clothing suspended from a waist-band.

Symptoms.—These seem to depend chiefly on the state of the nervous system. There may, indeed, be few or no symptoms, or there may be a dragging sensation in the abdomen, vomiting, constipation, and colic. The stomach, colon, and liver may be displaced downwards. The spleen and kidneys may be palpable. Serious symptoms occasionally result from kinking of bloodvessels or canals—*e.g.*, the ureter.

Treatment.—In this condition tight-lacing must be forbidden, and the clothing must be suspended from the

shoulders. Dyspepsia and constipation must be attended to. The neurasthenic condition requires careful attention, and if other measures fail, a course of Weir Mitchell's treatment should be resorted to. Occasionally it may be desirable that the surgeon should open the abdomen and anchor one of the displaced organs (*e.g.*, a kidney) in its normal situation by sutures.

xviii. Infarction of the Bowel.

This may result from blocking of the mesenteric vessels by thrombosis or embolism. In a large proportion of cases there is disease of the heart or of the abdominal aorta. The principal symptoms are abdominal pain and distension, vomiting, diarrhœa, and sometimes bloody stools. Diagnosis is of some importance, since in a certain proportion of cases resection of the damaged bowel is practicable.

xix. Intestinal Sand.

Sand or fine gravel may be present in the stools in large quantity and has various sources. It may consist of minute biliary calculi, or of the seeds of fruits, or of masses of vegetable cells. The colour varies from pale brown to deep red. The patients are frequently neurotic, and suffer from colitis. The passage of the sand may give rise to pain in the colon and rectum.

Tuberculosis and Syphilis of the intestine are described at pages 128 and 148 respectively.

DISEASES OF THE LIVER.

i. Jaundice

(Icterus).

This consists in a staining of the skin and mucous membranes by the bile-pigment. There are two varieties :

(1) Obstructive Jaundice (formerly called *hepatogenous* jaundice) results from obstruction of the biliary passages, whether the obstruction involve the large ducts or numerous minute intrahepatic channels. When the bile cannot escape into the duodenum, it is absorbed by the bloodvessels and

lymphatics in the walls of the biliary passages, and thus circulates in the blood. Obstruction may be brought about in many ways, as by gallstones or parasites in the ducts; inflammatory swelling with excess of secretion at the papilla in connection with catarrh of the duodenum; compression of the large ducts by tumours of neighbouring viscera, or by enlarged glands in the portal fissure; or compression of the minute ducts by tumours in, or cirrhosis of, the liver itself.

In ordinary cases, the skin is rendered yellow, but in severe cases it may be dark green ('black jaundice'). The abnormal tint is well seen in the conjunctiva, but it is important not to mistake for jaundice the *pinguecula* or yellowish spot due to thickened tissue, and formerly erroneously regarded as fat, which is present in many individuals.

Bile is present in the urine, and confers upon it a brownish colour combined with a varying depth of green. It can be recognised by Gmelin's test for bile-pigment. A few drops of the urine and a similar quantity of fuming nitric acid are poured, at a little distance from one another, upon a white porcelain surface, and then allowed to run together. When they meet, a play of colours is produced—green, violet, yellow, etc. Or the urine may be floated upon nitric acid in a test-tube; if bile is present, the layer of contact becomes green. Another method is to dilute the urine in a test-tube to get rid of the dark colour, and then add liquor iodi diluted with water to 1 in 10, or tincture of iodine diluted with rectified spirit to 1 in 3. Bile-pigment yields a green colour where the fluids join, owing, as before, to the oxidation of bilirubin into biliverdin. Pettenkofer's test for the bile acids is not applicable to urine directly. Bile is also present in the perspiration and stains the linen. Most of the other secretions are not as a rule stained. In cases that have died with jaundice, effusions in the serous cavities may show well-marked staining.

The absence of bile from the intestine causes a change in the stools. These are white and pasty, or claylike, and have a very offensive odour. Constipation is the rule, but sometimes there is diarrhœa. There may be flatulent dyspepsia. Fat is not well digested. There is sometimes a

bitter taste in the mouth. In addition to the yellowness of the skin, there is often severe itching (*pruritus*), and sometimes urticaria. In very chronic cases, xanthoma may develop.

Slowness of the pulse (*bradycardia*) is a well-known phenomenon of jaundice, and as it occurs chiefly in catarrhal cases, it is a favourable sign.

In chronic or severe jaundice, hæmorrhages may take place into the skin and from mucous surfaces.

Quite exceptionally, there is *xanthopsy*, so that white objects appear yellow.

Jaundiced persons often become irritable in temper. In severe toxæmic cases, and sometimes in prolonged obstructive cases, grave head symptoms develop—delirium, muscular tremors, coma, convulsions, a dry tongue, and other phenomena of the typhoid state. This condition probably results from the presence in the blood of toxic albuminous substances which in health are converted by the liver into urea. It is in no way related to the severity of the jaundice.

(2) Toxæmic Jaundice (formerly called *hæmatogenous* jaundice) is seen in yellow fever, pyæmia, pneumonia and other infectious fevers ; in poisoning by snake venom and by phosphorus and other mineral poisons ; and in some obscure infections of which acute yellow atrophy of the liver may be quoted as an example.

The jaundice is usually slight in this variety, whereas the constitutional symptoms are often very severe.

Toxæmic jaundice is due to the presence of toxic substances in the blood, which either cause excessive destruction of red corpuscles, or act upon the liver itself. The older view was that the jaundice is hæmatogenous (due to blood destruction with production of bile-pigment in the blood), but Hunter holds that this variety is as much obstructive as the other (*i.e.*, it is hepatogenous, the liver-cell being the seat of formation of bile-pigment). He believes that the toxins cause (1) increased destruction of blood with increased supply of hæmoglobin to the liver ; and (2) catarrh of the smaller bile-ducts with increased viscidity of the bile, which retards or even for a time arrests its flow, so that absorption

takes place. The difficulty with regard to this theory is that the stools may have the usual depth of colour, and there may be no trace of bile in the urine.

Treatment.—As jaundice is a symptom, the treatment is chiefly that of its cause. But it is not always possible to detect the cause immediately, nor desirable to postpone all treatment. Jaundice itself constitutes no reason why the patient should not take outdoor exercise if he is well clad. The diet should be light, and a mercurial purge should be given at the outset. An alkaline bitter tonic may be given before meals if the tongue is furred. If the latter is clean, dilute nitrohydrochloric acid should be given in combination with taraxacum. Nitrate of pilocarpine may be injected hypodermically to relieve severe itching, and warm baths may be employed for the same end. Or the skin may be sponged with liquor carbonis detergens diluted to the extent of 1 drachm in 1 pint of warm water.

ii. Icterus Neonatorum.

The majority of children present a certain amount of jaundice in the first week after birth. It usually appears about the second or third day, and disappears within a fortnight. In exceptional instances, jaundice arises in new-born children through obstruction of the bile-ducts, syphilitic disease of the liver, or septic infection of the umbilical cord.

iii. Acute Yellow Atrophy of the Liver (Malignant Jaundice. Icterus Gravis).

Etiology.—This is a rare disease which affects females more frequently than males, and is most common in the third decade of life. Many of the women who suffer are pregnant. Strong emotion seems to favour its occurrence, and other possible predisposing factors are syphilis, alcoholism, bad hygiene, and cirrhosis of the liver.

Morbid Anatomy.—The liver is flabby and greatly reduced in size. Its capsule is wrinkled. There is no bile in the gall-bladder or bile-ducts. On section of the liver, patches of different colour are seen, some being bright yellow, others

dark red, and others possibly with the usual colour of liver tissue. The yellow patches represent the earlier stage of the disease, and show under the microscope granular débris, leucin, tyrosin, and Charcot-Leyden crystals, with clumps of granular and atrophied liver cells. The red patches are firmer, and owe their colour to the absorption of the hepatic cells and distension of bloodvessels; in these areas there may be some increase of fibrous tissue.

Fatty or granular degeneration is also found in the epithelium of the kidneys and gastric glands, and in the cardiac muscle. The spleen is usually enlarged and soft, and hæmorrhages may be present in various parts of the body.

Pathology.—The jaundice has been attributed to catarrh of the finer bile-ducts resulting from the action of a toxin, but the toxin itself and the organism which produces it are as yet unknown.

Symptoms.—The severe symptoms may set in suddenly or may be preceded for days or weeks by slight jaundice of an apparently simple kind, and possibly also by some disturbance of the stomach and bowels. The more urgent symptoms include headache followed by delirium, tremor, convulsions, coma, a dry tongue, and a rapid pulse. There is usually no fever, unless just before death. The bowels are constipated, and the stools are in most cases devoid of bile. They may after a time contain blood. Vomiting is troublesome, and the vomited matter may contain blood or coffee-ground material.

The urine is normal in quantity, and is bile-stained. It frequently contains albumen and tube-casts. It is deficient in urea, but often contains leucin and tyrosin. According to one view, these are proteids which are evolved in the intestine, and are transformed into urea, under normal conditions, by the liver; but in this disease the liver is unable to discharge that function, so that leucin and tyrosin pass into the circulation and appear in the urine. The urea excretion is consequently reduced. Another view is that these two bodies are derived from the hepatic cells as a result of their destruction. Leucin takes the form of yellowish-brown highly refracting globular masses. Tyrosin forms colourless

sheaves of very fine needles. It may be necessary to concentrate the urine by evaporation to detect these two bodies.

The liver is tender, and can be observed to diminish in size with each succeeding day. The spleen at the same time enlarges, and various hæmorrhages occur, *e.g.*, petechiæ, epistaxis, and hæmatemesis.

Diagnosis.—This is based on the combination of vomiting and jaundice with progressive shrinking of the liver and severe nervous symptoms. In jaundice of a simple kind, the area of hepatic dulness may be greatly reduced, probably through the liver being overlapped by air-distended colon. The premonitory stage of acute atrophy cannot be distinguished from catarrhal jaundice.

The severe symptoms closely resemble those of *phosphorus poisoning;* but in the latter case there may be a history of swallowing phosphorus, phosphorus may be found in the vomited matter, gastric symptoms may be more marked, the urea excretion may be considerable, and finally the liver is large. Moreover, after death from phosphorus poisoning, the liver is found to be large and to contain much fat—in both points differing from the liver of acute yellow atrophy.

The presence of leucin and tyrosin in the urine is not peculiar to acute yellow atrophy.

Prognosis.—Only a few cases recover.

Treatment.—This is symptomatic. Purgatives should be given at first. Then intestinal antiseptics may be tried, such as salol, β-naphthol or bismuth salicylate. Hypodermic or intravenous saline injections may also be given, to aid in the elimination of poisonous substances.

iv. Cirrhosis of the Liver

(Chronic Interstitial Hepatitis).

Cirrhosis[1] of the liver is characterised by overgrowth of the interstitial tissue with destruction of the hepatic cells.

[1] The term 'cirrhosis' was applied to this disease by Laennec on account of the yellow colour of the liver. Its meaning has since been extended so as to include a similar pathological process, viz., overgrowth of interstitial tissue and pressure atrophy of parenchymatous elements, in whatever organ or tissue it may occur.

(1) In *portal* cirrhosis the morbid process starts from the branches of the portal vein in the liver.

(2) In *biliary* cirrhosis the starting-point is in the bile-ducts, and of this type two varieties have been distinguished, viz., (i.) hypertrophic biliary cirrhosis, which will be described in detail hereafter; and (ii.) obstructive biliary cirrhosis which was supposed to result from obstruction to the escape of bile. It is now believed, however, that so long as obstructed bile-ducts remain aseptic, no fibrosis takes place, but only local atrophy and destruction of the hepatic cells.

(3) An *intercellular* type of hepatic cirrhosis is recognised as particularly characteristic of inherited syphilis.

Alcoholic Cirrhosis (*common*, *atrophic*, *multilobular* or *portal cirrhosis*; *hobnailed* or *gin-drinker's liver*).—This disease is almost invariably the result of chronic excess in alcohol. It is induced by strong spirits more readily than by malt liquors. It is more common in men than in women, and the patient is usually at the prime of life. Children, however, may suffer, and of these some at least are alcoholic. Various specific fevers, and organisms absorbed from the bowel, have been suggested as causes, but these are doubtful.

Morbid Anatomy.—The fibrous tissue of the liver is greatly increased, and the hepatic cells are atrophied. The new fibrous tissue is of course preceded by a stage of round-cell infiltration. The organ is firm, and, when cut into, presents gray strands of fibrous tissue which extend from that in which the portal vessels are situated, and surround groups of hepatic lobules (*multilobular* cirrhosis). These lobules are yellow from bile-staining, and perhaps from fatty change. The groups of lobules projecting on the surface give rise to the *hobnailed* condition. The size of the organ as a whole varies much in different cases; it may be diminished (*atrophic* cirrhosis), or normal, or increased. The enlarged cirrhotic liver is sometimes very fatty (*fatty* cirrhosis).

The contraction of the new-formed fibrous tissue strangles the fine portal vessels, and thus leads to great obstruction in the portal system. One result of this is enlargement of the anastomoses between the portal and general venous systems. Of the numerous vessels of this class there may be specially

mentioned those connecting the œsophageal and gastric veins, those connecting the hæmorrhoidal tributaries of the inferior mesenteric and internal iliac veins, and those connecting the portal vessels in the round and suspensory ligaments of the liver with the epigastric and internal mammary vessels at the umbilicus.

The hepatic cells which still survive the pressure of the new fibrous tissue are atrophied, and sometimes fatty and pigmented. There is sometimes an appearance suggesting a new formation of bile-ducts, in the form of double rows of cubical epithelium, but the significance of these is still uncertain.

Other changes are chronic catarrh of the digestive tract, enlargement of the spleen, ascites, and often tuberculosis of the peritoneum, lungs, or other organs. Slight chronic peritonitis is common.

Pathology.—The poison which causes this form of hepatic cirrhosis reaches the liver by the portal vessels. It is natural that alcohol should be generally regarded as the cause of the fibrous tissue overgrowth, but it has been suggested that the influence of alcohol is indirect, viz., by causing catarrh of the stomach and bowels, and thus leading to the formation of abnormal products of digestion, which, being taken up by the portal vessels, irritate the liver. Other theories which have been proposed are too doubtful to require mention here.

Symptoms.—Cirrhosis of the liver may give rise to no symptoms, provided that the collateral circulation is sufficiently developed. The condition is often found after death when it was unsuspected during life. The symptoms are due partly to the chronic alcoholism and gastro-intestinal catarrh, partly to portal obstruction, and partly to a toxic state which may ultimately supervene. The patient has often the look of a drinker with a dirty skin, red nose, and dilated veins on the face. The tongue is furred, the appetite poor, the bowels usually costive but at times loose. There is sickness in the mornings. Hæmatemesis, melæna, and piles are common. The spleen is enlarged, and the superficial abdominal veins are sometimes distended. The urine is con-

centrated. Jaundice is usually slight or absent altogether. The liver may be enlarged and tender, or may be contracted. The large livers tend to be less nodular, and to be more often associated with absence of symptoms than livers which are reduced in size. Ascites and œdema of the feet are common, and the former may disturb the cardiac action and embarrass respiration. The temperature is occasionally subfebrile. In a case of enlarged cirrhotic and fatty liver which I had recently the opportunity of examining, the leading clinical feature was the occurrence from time to time of paroxysms of severe fever separated by intervals of very fair health.

Toxic symptoms, such as drowsiness, coma, convulsions, and delirium may set in at any time, and are of grave import. They are probably due to advanced atrophy of the liver cells rendering the organ unable to transform into urea some of the more poisonous antecedents of that body.

Diagnosis.—The most important points are the ascites, slight jaundice, hæmatemesis, dyspepsia, and history of drinking, sometimes with enlargement of the liver.

Prognosis.—This is unfavourable, but if head symptoms and ascites are absent, and other symptoms are slight, the patient may survive for years. Even head symptoms may be recovered from. Ascites and fever are usually of evil omen. Death may result from toxæmia, hæmatemesis, or tuberculosis of the peritoneum, lungs, or other organs.

Treatment.—Alcohol must be forbidden. The diet should be light and nourishing. Small doses of mercury and alkaline bitters should be given to improve the state of the digestive tract. An alkaline and bitter mineral water should be given in the mornings. Once the tongue is clean, nitro-hydrochloric acid may be prescribed in combination with nux vomica. When ascites is present, paracentesis should be performed. Hale White recommends copaiba resin (15 grains four times daily) as often removing a large accumulation of fluid from the peritoneum in this disease. The Talma-Morison operation, and the slighter operation known as *epiplopexy* or *omentopexy*, aim at increasing the anastomoses between the portal and systemic vessels by

causing adhesions to develop between the liver and diaphragm in the one case, and the great omentum and anterior abdominal wall in the other.

When toxic symptoms supervene, purgation, diuresis and the intravenous injection of saline solution are indicated.

Hypertrophic Cirrhosis (*unilobular cirrhosis, hypertrophic biliary cirrhosis, Hanot's disease*).

Etiology.—Males suffer more than females. Young adults and children are most commonly affected. Several members of a family may be attacked. Alcohol is not the cause, and, indeed, no undoubted cause is known.

Morbid Anatomy.—The liver is enlarged, firm, and smooth or finely granular. The connective tissue is increased around the individual lobules (*unilobular* cirrhosis), and may invade the lobules (*intralobular* or *intercellular* cirrhosis). In cases of long standing there is usually a secondary portal cirrhosis, due probably to poisons generated in the spleen and reaching the liver by the portal vein (Rolleston). The hepatic cells do not suffer so much as in alcoholic cirrhosis. There is proliferation of the fine bile-ducts. There is no obstruction of the large ducts. The spleen is greatly enlarged. The alimentary canal is free from catarrh. The liver is sometimes adherent to the diaphragm as a result of perihepatitis.

Pathology.—The inflammatory changes start from the bile-ducts, but whether these are due to upward extension from the duodenum, or to downward extension from the fine ducts—which have become inflamed through excreting a poison brought to them by the hepatic artery—is a matter of discussion. A microbic theory has been advanced, but is not yet proved.

Symptoms.—Jaundice is early and persistent, but not extreme. It is due to catarrh of the fine ducts, but the obstruction is not complete, since there is bile in the stools. The urine contains bile. The liver is much enlarged, smooth and tender. The spleen is enlarged and hard. The enlargements of the liver and spleen give rise to enlargement of the abdomen, but engorgement of the surface veins and ascites are quite exceptional.

The disease follows a chronic course, and the general

nutrition becomes much impaired. Hæmorrhages from the mucous membranes may occur now and then. Paroxysmal exacerbations of the jaundice are observed from time to time, frequently in association with attacks of abdominal pain and fever. The patient may die from hæmorrhage, or from exhaustion, perhaps after portal cirrhosis has been superadded to the original biliary type. Or the severe head symptoms of acute yellow atrophy may supervene and lead to the fatal issue, or death may be due to some other acute infection.

Diagnosis.—The persistent jaundice, with bile in the stools, the smooth enlargement of the liver, the splenic enlargement, the early age and the absence of alcoholism, are the characteristic features of the disease. The absence of ascites and of enlarged veins on the abdomen is also significant. The marked splenic enlargement, and the absence of colic, distinguish the disease from an attack of gallstones. In jaundice from obstruction of the common duct, bile is absent from the stools.

Prognosis.—The disease ends fatally after an average duration of about five years (from one to ten).

Treatment.—This is similar to that recommended for alcoholic cirrhosis.

v. Abscess of the Liver.

Abscess of the liver assumes various forms, and results from infection which may reach the organ by way of the hepatic artery as in general pyæmia, or by way of the portal vein as in ulceration of the intestine. Suppuration may also take place in the liver in consequence of infection by way of the gall-ducts, but this is described among the diseases of the biliary passages.

Of hepatic abscesses which result from infection by the bloodvessels, two principal types are distinguished, the *pyæmic* and the *tropical*.

Pyæmic Abscesses are multiple and of small size.

Etiology.—The infection usually comes from some part which is drained by the portal system of veins. Such abscesses, therefore, may follow ulceration of the stomach, intestines or bile-ducts ; operations for piles ; or abscesses

of organs connected with the portal system. Sometimes, however, abscesses in the liver are part of a general pyæmia, the organisms having passed through the pulmonary capillaries and hepatic artery.

Morbid Anatomy.—The liver is uniformly enlarged, and is studded with abscesses which are often of the size of peas, but which by coalescence may attain much larger dimensions. Some of these abscesses may be found to communicate with branches of the portal vein. There may be thrombosis and suppuration of the portal veins (*suppurative pylephlebitis*).

Symptoms.—If the affection of the liver is but part of a general pyæmia, there may be no symptoms indicating involvement of that organ. In portal pyæmia, there are general septic symptoms, such as hectic fever, sweating, and a dry tongue. The bowels are irregular. The liver is enlarged and tender, and there may be jaundice.

Prognosis.—Death occurs within a few weeks. Recovery is almost unknown.

Treatment is purely symptomatic.

The Tropical Abscess is generally large and solitary, but occasionally two or three such abscesses are present simultaneously.

Etiology.—In a large proportion of cases, the patients have suffered from amœbic dysentery.[1] Residence in a hot climate, excesses in eating and drinking, idle habits and defective sanitary conditions predispose to the disease. A chill, a drinking bout, or a blow on the liver may be the exciting cause. Men suffer more than women. Children are not liable. In rare cases, a large solitary abscess of the liver develops in individuals who have never been out of Britain. Ulcerative colitis, blows on the liver, foreign bodies, or suppuration in the neighbourhood may possibly account for such cases.

Cultures from the abscess are usually sterile. The disease is generally attributable to the *Amœba dysenteriæ ;* but there may be a secondary infection with such organisms as the *Staphylococcus aureus*, *Streptococcus*, and *Bacillus coli*.

[1] Abscess of the liver is rare in bacillary dysentery.

Morbid Anatomy.—In the formation of the abscess, a grayish patch appears which is surrounded by a zone of congestion. Softening beginning in the centre of the gray area gives rise to the abscess. The latter has ragged walls, and may ultimately contain several pints of fluid. Little abscesses may form around it, and burst into the large abscess. The contents are usually of a chocolate colour and somewhat thick, but they may resemble ordinary pus. They may include masses of necrotic liver tissue, but they consist largely of granular and fatty débris, with amœbæ, a few cells, and possibly Charcot-Leyden crystals. There is but little trace of ordinary suppuration, unless there has been a secondary infection with pyogenic organisms. The most common situation of a solitary abscess is near the back of the right lobe, but it may be in any part. Numerous abscesses may be present. The liver as a whole is somewhat enlarged.

Symptoms.—These are sometimes acute, but in most cases are insidious. There is slight malaise, with an irregular temperature, at times normal, at other times subfebrile, and, again, highly febrile. There is profuse sweating, especially during sleep. The spirits are depressed. The bowels are often loose, but sometimes constipated. Distinct jaundice is quite exceptional. The patient usually lies on his back or right side. There is sometimes a short cough, and in some cases vomiting. There is pain with tenderness in the hepatic region. Pain in the right shoulder is a common symptom, and suggests that the abscess is near the diaphragm. There may be bulging in the region of the liver in front, or percussion at the back may reveal an enlargement of that organ upwards and to the right, even though there is no downward enlargement. The disease causes progressive emaciation and weakness.

The abscess usually grows till it reaches the surface of the liver, and then, after causing adhesion of the serous surfaces, ruptures into the right lung, less commonly into the digestive tract, still less commonly on the surface of the body, occasionally into other parts (pericardium, etc.). Rupture may give rise to important symptoms. Thus, in the case of rupture into the lung, there will be cough, expectoration of

blood and chocolate-coloured material, percussion dulness on the right side, and œdema of the chest-wall from empyema. Rupture into the stomach may cause vomiting of material from the abscess. Rupture into the bowel may be followed by the evacuation of chocolate-coloured matter.

Tropical abscess may run a latent course until rupture takes place.

Diagnosis.—If hepatic abscess is suspected in a patient who has been abroad and has had dysentery, and who is losing ground through persistent fever and malaise, an exploratory puncture should be made (under an anæsthetic) with a medium-sized aspirator needle. According to Manson this trifling operation is infinitely less dangerous than procrastination, and is often curative by arresting a hepatitis before it has reached the stage of abscess. If there are no localising signs, the needle should be inserted in the seventh or eighth space in the anterior axillary line ; if no pus is found, it should then be inserted below the ribs slightly internal to the nipple line ; and if this fails, a third puncture should be made well below the lung in the line of the scapular angle.

Prognosis.—If the abscess is single, recognised in time, and properly treated by incision and drainage, half the cases recover. In rare instances, it dries in spontaneously and becomes obsolete. If it is allowed to go on to rupture, death usually follows, sometimes by very gradual exhaustion. Recovery may, however, take place after rupture into the lung or digestive tract. The duration of the disease varies from weeks to years.

Treatment.—The abscess should be opened with strict aseptic precautions, and thoroughly drained. If rupture has already taken place into the lung, stomach or intestine, an operation should be performed only if the purulent discharge, fever and other symptoms continue serious, so that the patient is manifestly losing ground. If the indications are doubtful, Manson recommends a change of air as being sometimes of great benefit in promoting healing. Several abscesses may have to be treated one after the other.

If the case is seen in the stage of hepatitis, before an abscess has actually formed, rest in bed and low diet are the

indications. Unless the bowels are loose, saline laxatives should be administered. Large poultices should be applied over the liver, and ipecacuanha should be employed as for dysentery. Chloride of ammonium is another drug of some value.

vi. Fatty Liver.

In this condition the hepatic cells contain an excess of fat. This may be due (1) to infiltration or accumulation, in which case fat is added to the normal cell contents; or (2) to degeneration, in which fat replaces some of the normal albuminous contents; or (3) to a combination of these two conditions, the persistent overloading with fat ultimately inducing degeneration.

Etiology.—Accumulation takes place in obesity, but in the long-run degeneration may be added. Acute degeneration takes place in phosphorus poisoning, and to a less extent in acute yellow atrophy. Fatty liver is also met with in chronic alcoholism, and in phthisis and other wasting diseases. In alcoholism, it is probably due both to excessive accumulation and to deficient oxidation. With regard to phthisis and other wasting diseases, there is a difference of opinion, some holding that the fat is infiltrated, and others that it results from degeneration. In phthisis the fat is specially deposited in the cells at the periphery of the lobules, as if it had been brought by the portal vessels.

Morbid Anatomy.—In a well-marked case of fatty liver, the organ is enlarged, smooth, soft, pale, greasy to the eye and greasy to the finger. It pits on pressure and floats in water.

Symptoms.—No symptoms can be referred to this condition. The liver is enlarged, smooth, soft, and free from pain and tenderness. There is no jaundice. Indeed, any symptoms from which the patient suffers, as well as the prognosis and treatment, will depend upon the cause of the fatty liver.

vii. Amyloid Degeneration.

(Albuminoid, Waxy or Lardaceous Degeneration).

Etiology.—This is part of a process tending to affect various organs of the body. It results from prolonged sup-

puration in connection with tuberculosis of the lungs or bones ; from syphilis, whether accompanied by suppuration or not, and from some less important causes. The introduction of the antiseptic system of surgery, and the diminished prevalence of phthisis, have naturally brought about a decline in the frequency of amyloid disease.

Morbid Anatomy.—The liver is heavy, firm and greatly enlarged. The cut surface has a waxy appearance. A weak solution of iodine renders the degenerated tissue mahogany red. Gentian violet stains the amyloid matter red and the normal tissue blue. The degeneration commences in the arteries and capillaries, and the swollen capillaries press upon the hepatic cells and cause them to become fatty and to undergo atrophy.

Symptoms.—Any symptoms that may be present are due to the cause of the degeneration or to amyloid disease elsewhere. The liver is enlarged, firm, smooth and painless. There is no jaundice. The spleen may be enlarged from a similar change. There may be albuminuria and dropsy from corresponding degeneration in the kidneys, and diarrhœa from similar changes in the intestinal villi. A cause will often be recognisable.

Prognosis.—This depends on the possibility of removing the cause, *e.g.*, bone disease of old standing ; but as a rule the outlook is very unfavourable.

Treatment.—This must be directed to the removal of the cause if possible, and to the improvement of the general condition by good food, tonics and change of air.

viii. Tumours of the Liver.

The principal growths are primary and secondary cancer, primary and secondary sarcoma, and angioma.

Primary Cancer is very uncommon. It has been estimated that about 4 per cent. of hepatic cancers are primary. Three varieties are described. In one, nodules are found throughout the liver as in secondary cancer ; one nodule, which may appear older, is primary, and the others are secondary. In a second type, the liver is much enlarged, owing to the presence of a single, large, well-defined tumour.

In the third variety, which is rare, the cancer cells are scattered all through the organ, and the fibrous tissue is greatly increased, so that the liver may be actually reduced in size.

Symptoms.—The disease occurs in middle-aged and elderly persons. Physical examination furnishes no means of distinguishing it from the much more common secondary cancer. According to Hale White, the course is more rapid, the jaundice less marked, and the stools less pale than in the secondary form.

Secondary Cancer is very common. A half of all the persons who die with malignant disease of any organ have secondary growths in the liver. The primary growth may be anywhere, but its usual seat is in the territory drained by the portal vein—*e.g.*, the stomach, gall-bladder, or head of the pancreas. It is probable that in many cases of so-called secondary tumours of the liver, the growths are really tertiary, the actual secondary growths being in lymph glands connected with the primary tumour. The disease occurs chiefly at and after middle life.

Morbid Anatomy.—The condition varies greatly. The secondary growths may be few or numerous, small or large, soft or firm. The presence of numerous large growths may cause great deformity of the liver. The organ may be several times the normal weight. The structure of the nodules will vary according to the primary growth. The larger nodules are apt to degenerate and soften in the centre, and this may be followed by absorption and shrinking, so that a depression or ' umbilication ' is produced in the centre of superficial nodules. Adhesions may exist between the liver and neighbouring structures, owing to extension of the growth from one organ to another. Dropsy, jaundice and other complications may be present.

Symptoms.—There are often pain and tenderness in the liver, and pain in the right shoulder. The liver may be felt to be enlarged downwards, and to have on its surface large nodules, which may or may not be umbilicated. A cancerous nodule is sometimes present at the umbilicus. Superficial lymph glands may be enlarged. The primary growth

may be accessible to examination. Jaundice, from compression of the bile-ducts, is present in about half the cases, and sometimes becomes very dark ('black jaundice').[1] Ascites, from compression of the portal vessels, or from involvement of the peritoneum, is not so common. Pyrexia is sometimes present.

Various symptoms from which the patient suffers may be due to the primary tumour, as in cancer of the stomach. The different phenomena of a cachectic state (a pale, earthy complexion, weakness, emaciation and ultimately dropsy) supervene, and death usually takes place in the course of some months.

Diagnosis.—Jaundice which persists for more than a few weeks in an individual who has reached or passed middle life should be regarded with grave suspicion. A nodular enlargement of the liver under such circumstances renders the diagnosis of cancer almost certain. The development of cachexia and the recognition of a primary tumour are also very important. It is desirable not to mistake cancerous infiltration of the great omentum for enlargement of the liver. It is sometimes difficult to distinguish between gall-stones and malignant disease in or near the gall-bladder; it must also be borne in mind that the two sometimes coexist. A distended gall-bladder should be distinguished by its position, size and shape from a growth in the liver. Various other enlargements of the liver may be excluded by the concomitant symptoms, or by the absence of symptoms.

Treatment is symptomatic.

SARCOMA.—Primary sarcoma is very rare. Secondary sarcoma may occur as a melano-sarcoma secondary to a similar growth in the eye or skin. It causes great enlargement of the liver, the nature of which may be surmised from the primary growth, and perhaps from secondary tumours in the lungs and skin, and from the presence of melanin in the urine.

ANGIOMA is rarely of clinical importance.

[1] Black jaundice may, however, result from any kind of obstruction of the bile-duct.

ix. Disturbances of the Circulation in the Liver.

Anæmia is of no clinical importance.

Active Hyperæmia of the liver has been suggested as the cause of various symptoms which occur in persons who eat and drink freely and take insufficient exercise. Thus there may be a sense of fulness and oppression in the hepatic region, a concentrated condition of the urine, headache and depression, a furred tongue, and slight yellowness of the conjunctivæ. These phenomena may well be accounted for by dyspepsia, and the poisoning of the liver by abnormal digestive products absorbed from the stomach and intestine.

The **treatment** consists in taking sufficient exercise, moderation in food and drink, restriction of stimulants, and an occasional dose of blue pill at night followed by an alkaline or bitter mineral water in the morning.

Passive Hyperæmia of the liver is an important consequence of disablement of the right side of the heart, due to emphysema, disease of the left heart, or some other cause. The liver is enlarged and firm, and on section has a marbled appearance (*nutmeg liver*) due to dilatation of the central vein, and relative pallor of the periphery of the lobules. The increasing distension of the lobular vessels leads to atrophy of the hepatic cells.

Symptoms.—There may be catarrh of the digestive tract, hæmatemesis, slight jaundice and even ascites. The liver is enlarged downwards, perhaps extending below the umbilicus, and is frequently tender.

Treatment.—Leeches should be applied over the liver. These not only remove blood from the engorged venous system, but may also give great relief to the local pain and tenderness. A mercurial purge should be given at the same time, and the bowels should be kept open by salines. The condition of the heart will of course demand immediate attention.

x. Perihepatitis.

Perihepatitis may be acute or chronic, general or localised. The *acute* form may be part of a general peritonitis. The *chronic* disease, more or less *localised*, may result from tuber-

cular, syphilitic, inflammatory or other processes involving the neighbouring part of the liver. Particular interest attaches to the *chronic general* form, which is characterised by opacity and great thickening of the whole capsule. The investigations of Fagge and Hale White suggest that the condition is part of a general simple chronic peritonitis, and that the cause is Bright's disease, the kidneys being usually granular. The capsule of the spleen is often involved in the same way as that of the liver (*perisplenitis*).

The liver is not enlarged, and jaundice is absent. The most important sign is ascites, which requires frequent tapping, as the fluid reaccumulates in consequence of the peritonitis. Along with the ascites and the absence of jaundice should be noted the presence of granular kidney. Hale White points out that the thickened omentum may be felt as a hard mass just above the umbilicus ; and that, as the fluid collects, percussion quickly becomes dull owing to the shortness of the chronically inflamed mesentery preventing the air-filled intestines from floating upwards and forwards.

Treatment.—Paracentesis must be performed as required.

DISEASES OF THE BILIARY PASSAGES AND GALL-BLADDER.

i. Simple Catarrh of the Bile-Ducts

(Simple Catarrhal Cholangitis. Catarrhal Jaundice).

Etiology.—Simple catarrh of the bile-ducts is a disorder of common occurrence in the first half of life. It is generally regarded as due to extension from the duodenum, though some writers think it commences in the fine ducts within the liver. The usual cause is irritation by abnormal products of digestion. The catarrh is associated as usual with excessive secretion of mucus, and this tends to obstruct the small orifice by which the duct opens into the duodenum. As the bile is secreted at very low pressure, absorption takes place and jaundice is produced.

Jaundice of a similar kind may follow a chill or a fright,

may accompany infectious diseases and may occur in epidemic form ; in most or all of these instances, catarrh is the probable explanation.

Symptoms.—The yellowness of the skin and conjunctivæ may be practically the only sign of the disease, or there may be slight dyspeptic symptoms with discomfort in the region of the liver. The stools are pale and offensive, the urine bilious (see Jaundice, p. 527), and the pulse sometimes slow. The liver is occasionally enlarged, and at the outset there may be elevation of temperature.

Diagnosis.—In young subjects previously healthy and with no signs of cachexia, the condition is clear. In middle-aged and elderly persons, particularly if the jaundice persists for months, the question of malignant disease must be carefully considered ; the latter will scarcely be disproved even if, for some time, there is no pain, emaciation or discoverable tumour.

Prognosis.—Recovery generally takes place after some weeks.

Treatment.—This consists chiefly in regulation of the bowels by alkaline aperient waters, light diet, an occasional dose of mercury with rhubarb and soda, and attention to any existing symptoms of dyspepsia. The patient does not require to keep in bed, unless there is considerable digestive disturbance. Sodium salicylate may be given, since it induces the liver to secrete more watery bile, and bile under higher pressure than in normal circumstances. Taraxacum has long been supposed to have a special influence on the liver, and may be prescribed in an alkaline tonic mixture.

ii. Infective Cholangitis.

Infective cholangitis is usually the result of gallstones, but it may be due to prolonged obstruction by malignant disease, worms, or foreign bodies ; or it may result from suppurative pylephlebitis. Microbes are the immediate cause.

Morbid Anatomy.—There is at first catarrh with lymphangitis. Later on there is suppuration, and the bile-ducts in the liver may be converted into abscess cavities. The infection may extend through their walls and cause inflammation

of the portal veins (*pylephlebitis*). There is often simultaneous suppuration of the gall-bladder.

Symptoms.—Attacks of intermittent fever with rigors, sweating and jaundice are the principal clinical evidence of the disease. These attacks of *hepatic intermittent fever* may occur from time to time for years without necessarily pointing to suppuration. But if the general symptoms are severe and continuous, and associated with enlargement and tenderness of the liver and of the gall-bladder, suppuration is probable.

Prognosis.—When suppuration has set in, the prognosis is very unfavourable.

Treatment.—This consists in removing the cause, which usually means opening and emptying the gall-bladder. This should be drained until the fever subsides.

iii. Obstruction of the Common Bile-Duct.

This may be due to gallstones, to cicatricial contraction following injury by gallstones, to round worms, or to malignant disease of the duct. More commonly it is due to pressure by some neighbouring tumour, especially of the pancreas or of lymph glands. The consequence is dilatation of the ducts, with persistent jaundice. The obstruction due to the presence of gallstones is commonly imperfect, so that some bile passes, and the jaundice varies from time to time ; in such cases, attacks of intermittent fever are common, and there is probably a history of hepatic colic. In obstruction due to pressure from outside, the jaundice is likely to persist or become deeper, and primary or secondary tumours may be recognisable.

Congenital obliteration of the bile-ducts is usually due, it would appear, to descending inflammation secondary to intra-uterine cirrhosis of the liver. It is characterised by severe obstructive jaundice, hæmorrhages, cholæmia, and a fatal issue. The liver is enlarged by a mixed cirrhosis, and the spleen also is enlarged.

iv. Cancer of the Bile-Ducts.

This is rarely primary. It generally involves the common bile-duct, and gives rise to dilatation of the ducts and gall-bladder, with early and persistent jaundice.

v. Catarrhal Cholecystitis

(Simple Catarrh of the Gall-bladder).

Simple catarrh of the gall-bladder often accompanies catarrh of the bile-ducts. The most important cause is digestive disturbance, and the most important result is the promotion of gallstone formation.

vi. Infective Cholecystitis.

Infective cholecystitis occurs in connection with gall-stones, and in enteric and other infectious fevers. It is favoured by simple catarrh. Pyogenic microbes are the immediate cause. Gallstones are a common result. The bladder becomes distended with a more or less purulent fluid (*empyema of the gall-bladder*).

The **symptoms** include pain and tenderness in the hepatic region, abdominal distension, and fever, but not as a rule jaundice.

In a few cases, described as *phlegmonous*, the infection spreads through the wall and causes perforation and peritonitis.

The **treatment** consists in prompt incision, evacuation and drainage of the gall-bladder.

vii. Cancer of the Gall-Bladder.

Cancer of the gall-bladder, when primary, is almost always associated with gallstones, a fact which explains why this growth is several times more common in women than in men. Cancer is doubtless often due to the irritation of gall-stones, but, on the other hand, it is probable that cancer, either by exciting catarrh, or by causing obstruction, favours the development of calculi. Secondary tumours grow in

the liver, so that the diagnosis may be difficult, but in most cases a tumour of the gall-bladder can be felt. A history of hepatic colic is important.

viii. Gallstones

(Cholelithiasis).

Etiology.—So far as is known, the first element in the production of gallstones is a catarrh of the biliary tract, whether due to the *Bacillus coli* or to some other agency. The catarrh on the one hand causes shedding of the epithelium, which may constitute a nucleus for a stone, and, on the other hand, is characterised by excessive secretion of mucus which is rich in cholesterin and bilirubin-calcium. The precipitation of the two substances last named gives rise to calculus. This may form in the ducts, but is much more common in the gall-bladder on account of the greater stagnation of bile. Any cause of catarrh in the gall-bladder or ducts may thus be regarded as a cause of gallstones. As enteric fever is occasionally complicated by cholecystitis, it may be regarded as an indirect cause of gallstones. Gallstones are several times as common in women as in men, and this arises from various circumstances, including tight-lacing, pregnancy, and sedentary habits, all of which prevent free movement of the diaphragm, and thus favour stagnation of bile. Gallstones seldom occur in subjects under thirty years of age ; but after that age, the tendency increases with each succeeding year of life. The influence of age is doubtless largely due to lack of exercise.

Characters of Gallstones.—Solitary gallstones are spherical or ovoid, and often consist of almost pure cholesterin, in which case they are nodular, amber-coloured, and glistening. They float in water. Multiple gallstones are more common, and are faceted from mutual pressure. They usually contain, in addition to cholesterin, a good deal of bile-pigment with calcium salts. These stones are grayish, brown or black. There may be only two or three, or there may be hundreds present. They may be of considerable size, or as small as grains of sand.

Symptoms.—Calculi may remain in the gall-bladder for an indefinite period without causing any trouble, and in most cases they never excite symptoms at all. Often, however, a stone passes into the ducts, and when passing through, or impacted in the biliary passages, it gives rise to important symptoms.

Hepatic colic (*biliary* or *gallstone colic*).—This is usually, but not invariably, produced when a stone enters the cystic duct or common bile-duct. The onset of an attack seems often to be related to the passage of chyme into the duodenum, since the pain frequently sets in three or four hours after a meal. It often begins at night. In a typical case, pain of intense severity sets in suddenly in the right hypochondrium, and radiates over the abdomen and into the back and shoulder. The patient writhes in his agony. There is severe and repeated vomiting, with sweating and a feeble pulse. At the outset there may be rigors and pyrexia. In half or more of the cases, jaundice supervenes, indicating that the stone has been in the common duct. There is great tenderness in the hypochondrium, and in some instances the liver is enlarged. The gall-bladder also may be enlarged and tender.

Some attacks are quite trifling. On the other hand, in a feeble subject, a severe attack may in rare cases cause fatal collapse.

The pain in an ordinary case passes off after some hours, but it may continue for days. It is brought to an end by the escape of the stone from the cystic duct into the common bile-duct.

The stone may not escape at once, or at all, into the duodenum, but may become impacted in the cystic duct or common duct.

Impaction in the cystic duct leads to distension of the gall-bladder, at first by bile, but ultimately by a clear mucous fluid. This may give rise to a large tumour projecting downwards from the liver. Later on, the gall-bladder may undergo atrophy. Or impaction may lead to acute inflammation, which is either simple, or more commonly suppurative. The empyema thus produced

may rupture, or may dry in and become infiltrated by lime-salts.

Impaction in the common duct is usually close to the duodenal orifice, the narrowest part of this duct. Jaundice is thus a common result, and may be intensified by secondary catarrh ; but if the stone remains movable behind the narrow orifice, there may be but little jaundice; or, again, there may be jaundice which varies greatly from time to time during a period of months or years. In such cases, where the stone acts like a ball-valve, *hepatic intermittent fever* may be well marked, each paroxysm being associated with hepatic pain and gastric disturbance, and followed by increase in the jaundice. The gall-bladder is seldom dilated (although when the common duct is obstructed from other causes than gallstones it is generally dilated). Impaction in this duct may further lead to cholangitis, which may be at first simple, but is very apt to become infective ; in the latter case there may or may not be suppuration. (See Cholangitis, p. 546).

Biliary fistula occasionally develops, a communication being established between the gall-bladder and the surface, through the skin ; or between the gall-bladder and the duodenum, colon, or some other canal or cavity. Fistulæ opening into the intestine may cause no symptoms.

Intestinal obstruction is another complication. The stone has generally ulcerated from the gall-bladder into the duodenum, and been arrested at the ileo-cæcal valve. The stone is usually passed spontaneously after a time, though the process may be painful.

Cancer of the gall-bladder is sometimes associated, as cause or effect, with gallstones. (See p. 548.)

Diagnosis.—The variability in the severity of attacks of hepatic colic should always be borne in mind, and also the further fact that gallstone colic generally occurs in repeated attacks. Moreover, repeated attacks of jaundice point to gallstones. For some weeks after a supposed attack these should be regularly looked for in the motions. The latter should be broken up with a stick on a sieve and carefully washed.

Treatment.—During an attack, the patient should have morphine hypodermically, and, if need be, chloroform by

inhalation till the morphine has time to act. A hot bath, hot fomentations, and an abundance of hot drinks are also to be recommended.

In the intervals the dietary must be carefully arranged, regular exercise should be taken, and the bowels should be slightly relaxed by the use every morning of Carlsbad water. Water should be drunk freely at bedtime and on rising, and also between meals, but not with food. Every endeavour should be made, by the help of alkalies, acids, bitters, and an occasional dose of mercury, to remove any catarrh of the digestive tract.

Various remedies have been employed to dissolve stones in the gall-bladder (*e.g.*, olive oil, or Durande's mixture of ether and turpentine), but there is no satisfactory evidence that such solution ever takes place, though in my own experience olive oil has appeared in some cases to be the means of preventing recurrence of the attacks. Frequently recurring attacks of colic, persistent jaundice due to impaction, and any evidence of acute inflammatory processes commencing in or around the gall-bladder may be regarded as indications for surgical intervention.

DISEASES OF THE PANCREAS.

i. General Considerations.

The internal secretion of the pancreas is produced by the islands of Langerhans, and has to do with the assimilation of carbohydrates. The external secretion is produced by the secreting cells of the pancreas, is poured into the duct of Wirsung, reaches the duodenum through the diverticulum of Vater, and acts as a powerful digestive juice. Disease of the pancreas may damage the islands of Langerhans, or the parenchyma of the gland, or both, and may thus interfere with the internal or with the external secretion, or with both.

In connection with the subject of diabetes, it was pointed out that experimental removal of the whole of the pancreas gives rise to *diabetes*. It is now known that any lesion which destroys the islands of Langerhans has the same effect. The

most common lesion of this kind, according to Moynihan, is a hyaline degeneration of the islands. In the terminal stage of *hæmochromatosis*, a rare disease in which pigmentation takes place in the skin and many other parts of the body, and in which there is multilobular cirrhosis of the liver and cirrhosis of the pancreas, the chronic interacinar pancreatitis ultimately involves the islands of Langerhans, and brings about diabetes (*bronzed diabetes*).

When the external secretion of the pancreas is entirely absent from the intestine, the stools are often quite distinctive, being grayish-white, pultaceous, and remarkably bulky. This peculiar colour is present even in cases where all the bile passes into the intestine ; it is uncertain whether this is because the fat in the stools is so abundant as to conceal the bile-pigment, or whether the latter, in the absence of the pancreatic juice, is absorbed from the intestine. But the presence of fat in the stools (*steatorrhœa*) is, by itself, no evidence of pancreatic disease, since it may occur in intestinal and mesenteric gland disease, and even in health. Absence of the pancreatic juice may allow of undigested proteid matter such as muscle fibre passing away in the evacuations (*azotorrhœa*) ; but so many fallacies may arise through changes in the patient's diet, and the presence of gastric or intestinal catarrh, that no great value can as yet be attached to this symptom. As Moynihan points out, however, steatorrhœa and azotorrhœa together, in the absence of jaundice, point strongly to pancreatic disease ; while a combination of steatorrhœa, azotorrhœa, diabetes, and epigastric tumour is conclusive evidence of such disease.

Sahli has introduced the use of glutoid capsules containing iodoform, which can withstand the action of the gastric juice for a considerable time, but which on reaching the intestine undergo digestion if pancreatic juice is present. In this way the iodoform is liberated, and the iodine reaction can be obtained in the saliva by testing with nitric acid and chloroform, the former setting free the iodine, which gives a pink colour to the latter. Salol may be used for this purpose in the same way as in testing the motor power of the stomach,

but it is obvious that each form of *Sahli's test* presupposes that the motor power of the stomach is nearly normal.

Since the common bile-duct and the duct of Wirsung join to form the ampulla or diverticulum of Vater, the short, common channel by which they discharge their contents into the duodenum, close to the head of the pancreas, it is not surprising that various diseases of the pancreas and its duct may give rise to *jaundice*. A group of symptoms which is very suggestive of primary cancer of the pancreas consists of deep-seated pain in the epigastrium or right hypochondrium, progressive emaciation, jaundice, and distension of the gall-bladder, but without a history of hepatic colic, and without definite signs of gastric carcinoma. According to 'Courvoisier's law,' persistent jaundice with distension of the gall-bladder depends upon cancer of the head of the pancreas.

ii. Pancreatitis.

Acute Hæmorrhagic Pancreatitis is a rare disease, and occurs chiefly in adult males. Alcoholism and cholelithiasis are among the possible causes. It may be produced experimentally by injecting artificial gastric juice or sterile bile into the duct of Wirsung, and it has been observed in man in a case where a gallstone obstructed the duodenal orifice of the ampulla of Vater in such a way that bile passed into the pancreatic duct.

Symptoms.—These include severe pain setting in suddenly in the region of the pancreas and followed by vomiting, abdominal distension and collapse, with death after a few days. The pancreas is found to be enlarged, with some of its cells necrosed and its tissue infiltrated with blood and round cells. In this, as in all kinds of pancreatic disease, there may be necrosis of fatty tissue in the pancreas, omentum and elsewhere. (See p. 557.)

Treatment.—As the intense pain and collapse are largely attributable to pressure upon the neighbouring plexuses, laparotomy is justifiable when this condition is suspected. Drainage of the gall-bladder cures or relieves some of the cases.

Suppurative Pancreatitis may be represented by a single abscess, or by multiple small abscesses. In addition to the general symptoms of suppuration, the existence of tumour in the pancreatic region is important in diagnosis.

Gangrenous Pancreatitis may follow inflammation of, or hæmorrhage into, the gland. The symptoms are the same as those of suppurative inflammation. Occasionally the necrosed organ is spontaneously evacuated with the fæces, and recovery takes place.

Chronic Pancreatitis is characterised by overgrowth of fibrous tissue and atrophy of the glandular elements. Among the causes are alcoholism ; extension from neighbouring lesions—*e.g.*, a gastric ulcer ; passive hyperæmia in heart disease ; and obstruction of the duct of Wirsung.

Two types of chronic interstitial pancreatitis are recognised. (1) In the *interlobular* variety the islands of Langerhans escape. Among the causes of this type is occlusion of the duct. (2) In the *interacinar* variety, the process is more diffuse, and the islands are involved.

The **symptoms** are not distinctive, but the condition is important because it may cause obstruction of the common bile-duct and jaundice, which may be wrongly attributed to cancer or gallstones ; and because it may be associated with diabetes (including the terminal diabetes of hæmochromatosis).

iii. Pancreatic Hæmorrhage.

This is a rare condition, and (apart from inflammation) of unknown causation. It is a cause of sudden death. In the midst of apparent health, the individual is seized with intense pain in the upper part of the abdomen, with nausea and vomiting. Collapse may lead to death in a few hours. The whole gland may be destroyed by the hæmorrhage, and the blood may spread into neighbouring parts.

Operation would be justifiable to relieve the pressure on nerve plexuses.

iv. Pancreatic Cysts.

The causes to which these cysts have been attributed are varied, and include blows on the abdomen and inflammatory conditions. They are occasionally retention cysts due to obstruction of the duct of Wirsung. Sometimes they may be part of a cystic adenoma of the gland. The cyst develops in the upper part of the abdomen, behind the stomach and transverse colon. It usually tends to advance towards the anterior abdominal wall between these two organs, but less commonly it is above or below both of them. The contents are often brownish or reddish in colour.

There may be no **symptoms** until the growth attains some size and weight. Pain and vomiting, however, may occur ; and jaundice, glycosuria, emaciation, and temporary decrease in the size of the cyst are occasional features. The cyst does not move with respiration, and is scarcely movable by the hand.

The **treatment** consists in evacuation or possibly extirpation of the cyst.

v. Tumours of the Pancreas.

The principal tumour is scirrhous cancer, and this usually involves the head of the gland. Secondary growths develop in the portal glands and the liver.

The **symptoms** include constant pain in the upper abdomen ; marked and persistent jaundice (due to pressure on the bile-ducts by the pancreatic tumour or by a secondary growth in the lymph glands) ; distension of the gall-bladder ; vomiting ; emaciation ; and occasionally steatorrhœa, glycosuria, or tumour in the epigastrium. (See p. 553.)

The **treatment** is mainly symptomatic, but a few cases have recovered after operation.

vi. Pancreatic Calculi.

Concretions consisting of lime salts are found in the pancreatic duct in rare instances. They may cause obstruction of the duct with dilatation, retention cysts or inflammation.

vii. Fat Necrosis.

This may occur in connection with any kind of pancreatic disease, and is sometimes observed when the pancreas appears to be healthy. The necrotic foci are often of about the size of a pin's head, but are occasionally much larger. The condition is most common in the peritoneum, especially near the pancreas, but it occurs in the pancreas itself, and occasionally elsewhere, *e.g.*, in the pericardial fat, subcutaneous fat, and bone-marrow. It is most common in the acute hæmorrhagic and necrotic varieties of pancreatitis, and is due to the action of the fat-splitting ferment of the pancreatic juice (lipase). The fat in the fat-cells is broken up into fatty acid and glycerin. The fatty acid is at first deposited in the cell in the form of acicular crystals, and then combines with the calcium salts to form a soap, while the glycerin is absorbed. Fat necrosis is due to lesions of the pancreas which allow the pancreatic secretion to escape from the duct into the tissues.

viii. Pancreatic Infantilism.

Bramwell has recorded a case of retarded development with chronic diarrhœa in which there was defect or absence of the pancreatic secretion, and in which great improvement took place under treatment by a glycerin extract of pancreas.

DISEASES OF THE PERITONEUM.

i. Ascites

(Hydroperitoneum. Dropsy of the Peritoneum).

This condition is characterised by the presence of an excess of serous fluid in the peritoneal cavity. The fluid has a pale straw colour, contains albumen, and has a specific gravity of about 1015. Ascites may be part of a general dropsy as in kidney disease, or in disablement of the right ventricle through disease of the lungs or left side of the heart. Or it may be due to obstruction of the portal vein or its branches by thrombosis, or by compression, the latter being

due, *e.g.*, to cirrhosis of the liver, tumours in the liver, or a tumour in the portal fissure. Or, again, it may be due to disease of the peritoneum itself, *e.g.*, inflammation, tuberculosis and cancer, though in these instances the fluid may be inflammatory rather than dropsical.

In rare cases, ascitic fluid is milky from the presence of chyle which has escaped from ruptured lacteal vessels (*chylous ascites*).

Physical Signs.—The abdomen is enlarged. In well-marked cases, if the patient is sitting up, it bulges forwards; if he is lying down, it bulges specially in the flanks. If it persists for some time, *lineæ albicantes* develop through over-stretching of the skin. The girth is of course increased. In severe cases, the upward pressure on the diaphragm may cause considerable interference with respiration.

The fluid gravitates to the dependent parts of the abdomen, so that as the patient lies on his back, percussion is dull in the flanks, and clear in the anterior or at least upper anterior part of the abdomen to which the intestines float. If the patient turns round on one side, the dulness will extend further towards the middle line from that side, whilst the side that is uppermost becomes clear to percussion.

Fluctuation as an evidence of ascites is obtained by placing one hand in contact with one side of the abdomen, and tapping the other side with one or two fingers of the other hand. To prevent a wave being transmitted along the anterior abdominal wall, it is well that an assistant should place the edge of his hand along the middle line of the abdomen.

Occasionally the method of *displacement* is of service. When a solid body, such as an enlarged liver or an abdominal tumour, is separated from the anterior abdominal wall by a layer of ascitic fluid, if the hand be placed over that solid body, and the fingers be then suddenly flexed, they displace the fluid and come suddenly against the hard object.

Diagnosis.—This is not always easy, and yet is of great importance with a view to treatment. A greatly *distended bladder* may simulate ascites; but the bladder causes distension and dull percussion in the lower anterior part of the

abdomen and not in the flanks. If there is any doubt, the catheter ought to be employed. An *ovarian cyst* causes dulness in front, while the flanks are clear; the tumour begins in one side of the abdomen; and vaginal examination may give valuable information.

Treatment.—When ascites is due to cardiac disability or to renal disease, purgatives are indicated. If these prove insufficient, or if at the first the abdominal dropsy causes great distress, the peritoneal cavity should be drained by Southey's tubes or by the aspirator. This method of treatment is also to be employed in the ascites of liver disease. If the effusion shows a strong tendency to recur, Barr's plan of injecting adrenalin chloride solution (2 or 3 drachms) may be tried.

ii. Acute General Peritonitis.

This disease is very often secondary to a lesion of an abdominal organ. Either inflammation extends from the viscus, or a hollow viscus ruptures and allows its contents, including microbes, to enter the peritoneal cavity. Appendicitis is one of the most common causes, and among the others are perforation of a gastric or intestinal ulcer, internal strangulation of the bowel, extension from salpingitis, and accidental and operative wounds penetrating the abdominal wall. Peritonitis occasionally supervenes in a late stage of Bright's disease.

Peritonitis is almost always due to microbes, and these usually reach the abdomen, either from the digestive tract, or by wounds from without. In the uncommon primary form, however, access may possibly be gained by the blood, or by the lymphatic system, or in some other way. The principal organisms are the *Streptococcus*, *Staphylococcus aureus*, *Pneumococcus*, and *Bacillus coli*. The tubercle bacillus is occasionally a cause of very acute peritonitis. In a large proportion of cases the *Streptococcus* is at fault. The *Bacillus coli* is found in nearly all perforative cases.

Morbid Anatomy.—The peritoneum is at first reddened, and as the intestine quickly becomes dilated, the redness is most marked in the spaces between the coils of intestine

where the pressure is least. Lymph (fibrin with leucocytes) is soon exuded,and glues the loops of bowel together, and in addition there may be serum in either large or small quantity. The serum may be so turbid as to suggest pus, or the exudation may be frankly purulent. Sometimes the effusion is hæmorrhagic. If perforation of the stomach or intestine has been the cause of the inflammation, gastric or intestinal contents may be present in the abdominal cavity.

Symptoms.—There may be shivering at first, but one of the most constant of the early symptoms is severe pain in the abdomen. In the perforation of enteric, one of the most important early signs is the abrupt change in the patient's appearance. The pain is at first general, but after a time it is often localised in the umbilical region. It is often accompanied by intense tenderness, and is aggravated by movement, *e.g.*, in coughing, vomiting, and breathing. The abdominal wall is more or less rigid, and the legs are often drawn up to relax the tension. Diaphragmatic breathing is in abeyance. Vomiting sets in early and causes much suffering. First the stomach contents are brought up, then yellow or green material, and ultimately, in some cases, brown matter with an odour suggestive of fæces. There is usually moderate, and in some cases high, fever, but in very severe cases there may be no elevation of temperature. The pulse is rapid, small and hard (wiry). The abdomen becomes distended owing to the great distension of the intestines, which results from paralysis of their walls. This excessive tympanitic distension of the abdomen is called *tympanites* or *meteorism*. Though percussion generally yields a tympanitic note over the abdomen, a dull note may be obtained in the flanks if the effusion is considerable ; and, indeed, the presence of free fluid may be recognisable as early as two hours after the onset. If the exudation glues together the loops of bowel, this dulness may not shift with changes of posture. Obliteration of the hepatic dulness is one of the recognised signs of rupture of an air-filled viscus into the peritoneum, but this sign may be due to flatulent distension without rupture. The bowels are usually but not always constipated. There may be difficulty with micturition.

The urine is concentrated and contains an abundance of indican (for tests, see Examination of the Urine). Collapse sets in quickly, with the pinched features, sharp nose, sunken eyes, and other elements of the Hippocratic face (*facies Hippocratica*). Death usually takes place from exhaustion in from three to six days. In cases which end fatally within forty-eight hours after operation, or after perforation, it is difficult to say how far shock, and how far rapid septic poisoning is the actual cause of death.

Diagnosis.—The phenomena already described (abdominal pain and tenderness, the *facies*, vomiting, meteorism and collapse) suffice for typical cases. The history or the present condition of the patient may make the occurrence of peritonitis intelligible (*e.g.*, in gastric ulcer, enteric fever, appendicitis, pelvic disease, etc.). In *colic*, the pain may be actually relieved by pressure, the temperature is undisturbed, and a cause (*e.g.*, lead poisoning) may be recognisable. Almost all the symptoms of peritonitis may be simulated by *hysteria*, and the course of the disease may alone permit of a distinction being made. In *intestinal obstruction* there is no fever ; the vomiting is more severe than in peritonitis, and is sometimes fæcal ; the constipation is more complete, and there is not so much tenderness and distension as in peritonitis.

Prognosis and Treatment.—In acute general peritonitis, the prognosis is practically hopeless unless surgical measures are adopted without delay. As a rule no opiate should be given until the question of operation has been considered. If this is decided against, absolute rest, rectal feeding, and opium in large doses are the principal indications. The opiate, by stilling the movements of the bowel, favours the development of adhesions. Some authorities, on the other hand, recommend saline purgatives. Ice may be sucked to allay thirst, and an ice bag or hot fomentations should be applied to relieve pain. In a strong subject, the fomentations may be preceded by leeching.

iii. Acute Localised Peritonitis.

Localised acute peritonitis occurs in various forms, of which the three following are the most important :

(1) *Subphrenic peritonitis* may be due to extension from the pleura, but is more commonly the result of disease of an abdominal viscus, and especially the stomach, intestine or liver. The inflammation often goes on to suppuration, and in most of the cases which follow perforation of the stomach or duodenum, the abscess contains air as well as pus (*subphrenic pyopneumothorax*). When the abscess results from disease of the liver, it is usually on the right side ; whereas if due to perforation of a gastric ulcer, it is generally on the left side, between the diaphragm and the left lobe of the liver. A subphrenic abscess closely simulates empyema, and if it contains air, the physical signs are suggestive of pyopneumothorax. The diaphragm is pushed up, the liver is depressed, and tympanitic percussion and amphoric phenomena are observable. The pre-existence of disease in an abdominal viscus and the previous absence of chest symptoms are important guides in diagnosis.

The treatment of these cases is surgical.

(2) *Appendicitis*, as has been already explained (p. 515), is really a peritonitis starting from the appendix. It may or may not be suppurative.

(3) *Pelvic peritonitis* is common in women. The Fallopian tubes, and less commonly the uterus, are the usual sources of infection.

iv. Chronic Peritonitis.

Etiology.—Chronic peritonitis may be either localised or general. The *localised* form may be met with as a thickening of the capsule of the liver (*perihepatitis*) or spleen (*perisplenitis*) ; or in connection with recent or old ulceration of the stomach or bowel ; or in connection with appendicitis. Some of these conditions may lead to the formation of bands under which a loop of intestine may slip and be strangled.

A more *general* inflammation may occasionally result from

acute or subacute peritonitis, or from alcoholism, Bright's disease or some other morbid blood state. Tubercle and cancer are common causes. Peritonitis may be part of a *polyorrhomenitis* (*polyserositis* or multiple inflammation of serous membranes), whether tubercular or due to some other agency.

Morbid Anatomy.—The principal changes are thickening and opacity of the peritoneum, with adhesions of the viscera through the agency of new-formed fibrous tissue. The omentum and mesentery may be shortened. Sometimes there is effusion, and this may be serous, sero-fibrinous, purulent or hæmorrhagic.

Symptoms.—These are not very distinctive, and, indeed, there may be no symptoms at all. There may be uneasiness or pain in the abdomen with constipation. Slight symptoms may be of value if there is a history of a preceding acute attack, or of some disease which might lead to peritonitis. There may be physical signs of effusion similar to those of ascites. Friction can sometimes be felt by the hand placed on the abdomen.

Prognosis.—This varies greatly according to the cause and extent of the disease.

Treatment.—Where possible, the cause should be removed. The general health must be carefully attended to, and the bowels should be regulated. If effusion is present in large quantity, it may be removed by paracentesis, and this operation may require to be repeated. Purulent collections must be treated by surgical methods.

v. Cancer of the Peritoneum.

Cancer is the most common tumour of the peritoneum, and is almost always secondary. It usually originates in the stomach or ovary. It is chiefly seen after middle life, and is more common in women than in men. Firm white nodules, sometimes umbilicated, are scattered all over the peritoneum, and may be so closely aggregated as to cause great thickening. Chronic peritonitis with hæmorrhagic effusion may be present.

The **diagnosis** is made during life by the cachexia, the abdominal pain and tenderness, the nodular thickening of the peritoneum, and the age of the patient. There may be evidence of tumour in the stomach, ovary, or elsewhere. The development of a nodule at the umbilicus, and the enlargement and hardening of a superficial lymph gland, are very significant.

SECTION VII

DISEASES OF THE KIDNEYS

1. EXAMINATION OF THE KIDNEYS.

The left kidney is generally situated half an inch or more higher than the right. The upper end of the left kidney is at the level of the eleventh dorsal spine. Its lower end is just below the level of the second lumbar spine, and is distant about 2 inches from the iliac crest. The hilus is about 2 inches from the middle line, and corresponds in level to the first lumbar spine. The colon passes vertically in front of the outer portion of each kidney.

If the kidneys are normal in size and situation, they cannot be examined by inspection, or even in most cases by palpation, unless the individual is greatly emaciated. It might be expected that percussion would reveal the situation of the lower part of the kidney with portions of its inner and outer borders, but in practice the results are of no great value under normal conditions.

If, however, the kidney is much enlarged, or if it is absent from its normal situation, these physical methods of examination may yield important information. Skiagraphy and an exploratory incision are other methods at our disposal. In the main, however, our knowledge of the state of the kidneys during life is derived from examination of the urine. Occasionally it is desirable to examine the urine secreted by a particular kidney, and this may be done by catheterising the ureter connected with that kidney, or by compressing the opposite ureter and so preventing the urine which it transmits from reaching the bladder. Luys's

'urine separator' is an apparatus which, when introduced into the bladder, divides that cavity into two lateral halves by means of a rubber partition. It also includes two catheters, each of which drains one half of the bladder, and thus transmits the urine from the corresponding kidney (see p. 573).

2. THE URINE.

Urine is a watery solution of nitrogenous waste products and certain salts. In 1,000 parts of normal urine there are about 955 parts of water and 45 of solids. Of these 45 parts, about 22 consist of urea, and 17 of alkaline and earthy salts. Most of these substances pre-exist in the blood and are separated from it by the kidneys; the water and mineral salts passing through the glomeruli, and the urea being removed by the epithelium lining the tubules. The uric acid, however, is perhaps actually formed in the kidneys. In disease the proportion of solids often deviates greatly from the normal.

Colour.

The urine in health is generally of a straw colour, but is paler if the quantity is large, and darker if the excretion is scanty. The urine is accordingly pale in diabetes mellitus, diabetes insipidus, granular kidney, amyloid kidney, and hysterical and other functional nervous disturbances. On the other hand, it is high-coloured in fevers, certain diseases of the liver, and profuse perspiration. In disease, however, the high colour may be due to the presence of abnormal pigments as well as to concentration of the normal colouring matter. Again, in renal disease, the urine may be pale, though the quantity is below the normal.

In health the colour depends chiefly upon a pigment called *urochrome*. The urine also contains *chromogens* or bodies which, when decomposed, give rise to derived pigments. Thus, *indican* (*potassium indoxyl sulphate*), which is derived from indol, a product of decomposition in the intestine, may become oxidised in decomposing urine and yield *indigo blue*. It is increased in peritonitis, obstruction of the small intes-

tine, wasting diseases, and conditions where there is increased putrefaction of proteids. It is recognised by mixing equal quantities of the urine and hydrochloric acid, and then adding drop by drop a saturated solution of chlorinated lime, and shaking until a blue colour appears. The colouring matter can be separated by shaking up with chloroform The chloroform on standing sinks to the bottom of the test-tube coloured blue, while the supernatant fluid is red or purplish. If the blue-coloured chloroform is afterwards evaporated, the blue pigment remains as a deposit. *Urobilin* is normally present only in traces, but may be much increased in disease, *e.g.*, in pernicious anæmia, giving the urine a dark reddish-brown colour. *Uroerythrin* is normally present in small quantity, and gives the colour to pink urates. *Hæmatoporphyrin* (which is hæmatin free from iron) is also present in small quantity under normal conditions, but is sometimes much increased—*e.g.*, in rheumatism, and after large doses of sulphonal. It then imparts a deep red colour to the urine. It does not give a definite reaction with guaiac, but is recognisable by the spectroscope. In cases of melanotic sarcoma, the urine may contain a chromogen which, on oxidation (*e.g.*, by nitric acid), or spontaneously on standing, is converted into *melanin*, rendering the urine black. Such urine gives with bromine water a yellow precipitate which gradually becomes darker. It also gives with strong ferric chloride solution a gray colour and a precipitate of phosphates and pigment which is soluble in excess.

Besides normal and pathological pigments and chromogens, the urine often contains colouring matters which are adventitious, and due to the presence of substances which, owing to abnormal circumstances, have been added to the urine. Thus, *blood* and *bile* impart their colours to the urine. *Hæmoglobin* and *chyle* may also be mentioned. Certain drugs affect it. For instance, *rhubarb*, *santonin* and *senna* may give it a yellow colour, which is changed into red by the addition of an alkali. *Methylene blue* renders the urine blue. *Carbolic acid* absorbed from dressings may render the urine dark green or black, the colour being due to the oxidation of hydrochinon and pyrocatechin.

Alkaptonuria is a rare condition of life-long duration, in which the urine, though of a natural colour when voided, becomes gradually darker on exposure, till it is reddish-brown, dark brown, or even black. This colour is due to the presence of dihydroxyphenylacetic acid. Urine of this kind reduces Fehling's solution ; but the fermentation, polarimetric, phenyl-hydrazin, and bismuth tests are all negative. Alkaptonuria appears to be associated in some instances with another very rare condition, viz., *ochronosis*, in which there is blackening of the cartilages, and it may be of the sclerotics and skin. The evidence at present seems to show that alkaptonuria is one cause, but not the only cause of ochronosis. Alkaptonuria may occur in several members of a family. It is harmless and usually congenital. It may be regarded as the expression of an anomalous form of proteid metabolism.

Odour.

This is almost characteristic, and is described as *urinous*. Urine which is alkaline from *fixed alkali* has a sweet odour. The presence of *ammonia* (volatile alkali) gives a distinctive odour. Decomposing urine may smell very offensively. Diabetic urine has a sweet odour, described as that of newly-mown hay. When such urine ferments, it acquires a distinctive odour suggestive of sour milk. In diabetic coma, the urine has, owing to the presence of *acetone*, an odour like that of chloroform. *Coffee*, *garlic* and *asparagus*, and various drugs, such as *rhubarb*, *cubebs*, *copaiba* and *sandal-wood*, confer their odour on the urine. The inhalation or internal administration of *turpentine* gives the urine an odour of violets.

Quantity.

In the normal adult, the urine excreted amounts to about 50 ounces daily. It is diminished (*oliguria*) when there is diarrhœa or free sweating, in general venous congestion, in cirrhosis of the liver, and in some forms of kidney disease. It may be suppressed altogether (*anuria*) in severe shock, in the collapse stage of cholera, and in connection with various

lesions of the urinary organs. It is increased in both forms of diabetes, in hysterical paroxysms, and in certain forms of kidney disease (especially the early stages of granular and amyloid kidney). Urine which is scanty is usually of high colour and of high density, but in some diseases of the urinary organs this is not the case.

Density.

The specific gravity is ascertained by means of the *urinometer*, and is normally about 1020. After free drinking of liquid, the density tends to fall as the quantity increases. In hysterical paroxysms, in some types of kidney disease (especially granular and amyloid), and in diabetes insipidus, the density is low. The density is high in diabetes mellitus, in febrile and hepatic disorders, and after profuse sweating. In a rough way the proportion of solids in any specimen of urine may be estimated by *Trapp's formula ; i.e.*, multiply the last two figures of the specific gravity by two, and the result represents the solids per 1000 of urine. *E.g.*, if the urine has a density of 1018, there are approximately 36 parts of solids per 1000 parts of urine.

Molecular Concentration.

Cryoscopy[1] has recently come into use in the investigation of the renal function, especially as a guide to surgeons who may propose to operate on a kidney. The method depends on the fact that the greater the number of molecules in a solution—the greater the molecular concentration—the lower is the freezing-point. Under normal circumstances human blood has a fixed freezing-point at 0·56° C. below that of distilled water ; this is recorded as — 0·56° C. In the case of urine, on the other hand, the freezing-point may vary under healthy circumstances from — 0·9° C. to — 2° C. When renal inadequacy leads to the retention in the blood of metabolic products, the freezing-point of the blood is lowered. If one kidney is able to compensate for disease in or loss of the other, the freezing-point of the blood remains

[1] Gr., κρύος, frost ; σκοπεῖν, to examine.

normal. If one kidney is diseased, the urine from that kidney (being less rich than normal in excretory products) will freeze at a higher temperature than the urine from the healthy and more active kidney.

Reaction.

This is usually acid, and depends chiefly upon the presence of acid sodium phosphate. The acidity is greatest in the morning and least after meals. Some hours after the heavier meals, the urine secreted by the kidneys may be alkaline for a time. In addition to food, alkalies and their salts render the urine alkaline, and the organic salts of sodium and potassium (acetates, bicarbonates, citrates, etc.) may be administered for this purpose for months at a time without doing harm. Five or six drachms may be given daily in divided doses. Whichever salt is selected, it is probably changed in the intestine into the carbonate before absorption. On the other hand, the internal administration of acids has very little influence in replacing an habitually alkaline by an acid reaction.

In some diseases, the urine tends to be alkaline from fixed alkali, *e.g.*, chlorosis, gastric ulcer, etc. In certain of these affections, the frequent loss of acid by vomiting accounts for the increased alkalinity of the blood.

A very important condition of the urine is alkalinity from volatile alkali, a condition which ought not to arise for many hours after the urine has been evacuated, but which occasionally supervenes before evacuation, as in many cases of cystitis. This ammoniacal decomposition is brought about by the agency of microbes, the urea taking up water and becoming converted into ammonium carbonate—

$$\underset{\text{urea}}{CO(NH_2)_2} + 2\underset{\text{water}}{H_2O} = \underset{\text{ammonium carbonate.}}{(NH_4)_2CO_3}$$

The ammonium salt is highly irritating to the mucous membrane and naturally aggravates the cystitis.

The reaction of urine is recognisable by its effect on litmus paper, and sometimes by the odour. If it is alkaline, red litmus paper becomes blue. If this is due to volatile alkali,

the blue colour will be lost on gentle warming over a spirit-lamp, whereas with fixed alkali the colour will be retained. Acid urine turns blue litmus red. The reaction can sometimes be judged by the eye ; *e.g.*, concentrated urine which throws down the common pink urates is always acid.

Urea.

Urea constitutes about 2 per cent. of the urine, or half the solids. About 500 grains are excreted daily by healthy men ; but as this is the principal form in which nitrogenous waste products are removed, a much smaller excretion may be looked for from patients who are kept on a diet poor in nitrogen.

Various instruments (*ureometers*) have been devised for the quantitative estimation of urea. Those which are most used decompose the urea and estimate it on the basis of the nitrogen evolved. The specimen examined should be taken from the quantity collected for the whole twenty-four hours. If albumen is present in the urine, it should first be removed by acidulation, boiling and filtering. In using the *instrument of Doremus* (sold by Southall), the hypobromite solution is poured into the apparatus until the whole of the vertical tube and half of the bulb are filled. The pipette is filled up to the mark with the urine to be tested, and is then cautiously introduced through the bulb into the lower part of the vertical tube. The rubber top of the pipette is slowly compressed till all the urine is driven out. The nitrogen evolved collects at the top, and displaces the liquid contents. The divisions on the tube indicate 0·001 gramme urea per c.c. of urine, and 1 grain urea per fluid ounce of urine.

In using *Gerrard's apparatus*, 5 c.c. of the urine are poured into the tube, and 25 c.c. of the hypobromite solution are put in the bottle. The tube is placed inside the bottle so that none of its contents are spilled. The other part of the apparatus is then filled with water, and the funnel is moved up or down till the level of water in the graduated tube is at zero. The bottle and the graduated glass are now firmly corked, and thus connected with one another by the tubing which passes through both corks. The bottle is then tilted

so as to allow the urine to escape from the tube into the solution in the bottle, with the result that decomposition takes place and nitrogen is evolved. The gas passes by the flexible tubing into the graduated glass, where it displaces the water downwards. After the water has come to rest, the funnel is lowered until the water is at the same level in funnel and graduated tube. The degrees of displacement are then read off as a percentage : 0·1 per cent. is nearly equal to 0·44 grain urea per fluid ounce of urine.

The hypobromite solution which is used to decompose the urea consists of caustic soda 10 grammes, and water 25 c.c., to which are added (properly speaking, just before use) 2·2 c.c. of bromine. For use with Gerrard's apparatus, this quantity of bromine can be obtained in a hermetically sealed tube, which is to be placed in the bottle along with 5 c.c. of urine and 25 c.c. of the caustic soda solution. After the apparatus is properly corked, the bromine tube is broken by a smart shake of the bottle.

The excretion of urea is augmented in fevers and in diabetes, and is diminished in Bright's disease. When the urine is concentrated, either by artificial means or to the extent naturally present in many cases of pneumonia, the addition of nitric acid yields a crystalline cake of *nitrate of urea*—a change which is accelerated by immersing the test-tube in cold water.

Drug-Tests of Renal Excretory Power.

The use of drugs for the purpose of estimating the degree of renal sufficiency has the advantage over the estimation of urea that a known quantity is introduced into the body at a definite time, so that delay in excretion can be detected, as well as diminution in the amount excreted as compared with normal conditions. Unfortunately, however, it has been found that the elimination of these drugs does not always keep pace with the elimination of the natural metabolic products, so that the usefulness of this method is very restricted.

One c.c. of a 5 per cent. watery solution of *methylene blue* may be injected into the gluteus maximus. Before the injection is made the solution should be filtered, and sterilised

by boiling, and the patient should empty his bladder. The urine is collected at intervals of a half, one, two, three, six, twelve or more hours, and tested for methylene blue. As this is at first excreted in the form of a colourless chromogen, acetic acid is added to the urine, and the latter is then heated; whereupon the development of a green or blue colour will reveal the presence of methylene blue. Within half an hour, however, under normal circumstances, the urine itself becomes pale green, and in the course of three or four hours a dark green colour is attained. This continues for some hours, and then gradually fades, the green tint passing away altogether in forty or fifty hours from the time when the drug was administered. It may be advisable to apply the methylene blue test in conjunction with the use of Luys's separator (pp. 565-566).

Four c.c. of a 4 per cent. solution of *indigo carmine* may be injected in the same way. It has the advantage that no treatment is required to display the colour in the urine. With healthy kidneys, the colour appears in the urine in about ten minutes, reaches its maximum in thirty minutes, and gradually disappears in the course of two or three hours.

One c.c. of a 1 per cent. solution of *phloridzin* (dissolved in water with the help of sodium carbonate), when injected as before, gives rise to glycosuria, which is recognised by the usual tests. This phenomenon persists for from two to four hours, and the amount of glucose normally excreted varies from $\frac{1}{2}$ to $2\frac{1}{2}$ grammes. It may be found in cases of acute and subacute parenchymatous nephritis that methylene blue is excreted while glucose is not.

Uric Acid (Lithic Acid, $C_5H_4N_4O_3$).

Uric acid is excreted to the extent of from 7 to 10 grains daily. Its importance is due chiefly to its great insolubility. It is present in the urine in combination with the alkalies. Under certain circumstances (one of which is high acidity) uric acid is deposited by healthy urine after standing for some time. The reddish crystals assume many shapes, such as squares, barrel-staves, rosettes, etc. They appear to the naked eye like grains of cayenne pepper lying at the bottom

of the glass, adhering to cracks and hairs, and floating on the surface of the urine. The murexide test may be used to identify uric acid chemically. A small quantity of the suspected matter is heated on a porcelain capsule with a drop of nitric acid, and allowed to cool. A drop of dilute ammonia is then added, and the purple red colour of murexide will appear if uric acid is present. In some diseases, such as leucocythæmia and pernicious anæmia, the excretion of uric acid is increased, but a much more important anomaly is the precipitation of uric acid before the urine has left the urinary tract. Uric acid is supposed to play an important part in the etiology of gout.

Urates (Lithates).

Amorphous or *mixed urates* are often precipitated when the urine cools in the form of a reddish or sometimes pale amorphous sediment (*lateritious* or *brick-dust deposit*). This is readily dissolved by heating the urine. Its significance is acidity and concentration, from whatever cause—*e.g.*, fever, hepatic disorders, or profuse perspiration. These urates are acid salts of potassium, sodium and ammonium, and have been alluded to in connection with gout (*q.v.*).

Ammonium urate occurs in the form of spheres and dumbbells in ammoniacal urine.

Sodium urate sometimes occurs in the form of hedgehog crystals in the febrile urine of children.

Chlorides.

Chlorides are present in the urine chiefly as sodium chloride, and are demonstrated by adding nitric acid, and if need be filtering, and then adding a solution of silver nitrate, which throws down the chlorides as a white curdy precipitate. The chlorides in the urine are diminished in febrile conditions, and are practically absent in many cases of pneumonia.

Phosphates.

Phosphates exist in the urine chiefly in combination with sodium and potassium, and are never then deposited. A smaller proportion is combined with the earthy bases, viz.,

calcium and magnesium, and these earthy phosphates tend to be deposited in alkaline and even in neutral or faintly acid urine. They may assume three forms: (1) *amorphous phosphate* of calcium, or *bone earth*, $Ca_3 2PO_4$; (2) *crystallised phosphate* of calcium, or *stellar phosphate*, $CaHPO_4 + 2H_2O$; (3) *triple* or *ammonio-magnesian phosphate*, $MgNH_4PO_4 + 6H_2O$. The three forms may be precipitated together, but much more commonly the first and third, which constitute the *mixed* or *secondary phosphates* ordinarily deposited by ammoniacal urine.

When alkaline or faintly acid urine is heated, the earthy phosphates often produce an opacity which is at once cleared away by the addition of a drop or two of acetic acid. The explanation of this phenomenon is still doubtful, though several theories have been proposed.

Stellar phosphate occurs in rods and stars; *triple phosphate* in triangular prisms with bevelled ends (coffin lids and knife rests), and sometimes in other forms; *amorphous phosphate* as a white sediment soluble in acid.

Phosphatic diabetes is a condition recognised by some writers, and characterised by polyuria, thirst, loss of flesh, and a great increase in the excretion of phosphates. The urine is not invariably free from sugar.

Calcium Oxalate.

Calcium oxalate constitutes the 'powdered wig deposit' which is sometimes seen on the top of a normal mucous sediment. The microscopic crystals are generally octahedra, and look very like square envelopes, but are sometimes dumb-bells or ovals. The urine is generally acid. Oxalic acid is almost constantly present in urine in small quantity, and is probably derived both from the food and from tissue metabolism.

The deposit frequently follows the taking of rhubarb as a vegetable. An occasional deposit of oxalate has no special significance, but if it is habitual and abundant, it suggests the possibility that a calculus may develop. Moreover, different authorities have recognised an *oxalic acid diathesis*, characterised by habitual oxaluria, together with general

nervousness, depression of spirits, pain in the back, and other symptoms of neurasthenia. It is not likely that these symptoms result from the oxaluria ; it is much more probable that both are due to dyspepsia, or some other common cause.

Sulphates.

Under normal conditions, sulphuric acid in the urine is mostly excreted in the form of sulphates of potassium, sodium and magnesium. A much smaller proportion is present in the form of aromatic sulphates, viz., sulphuric acid combined with one of the inorganic bases just mentioned, and also with an aromatic radicle such as phenol, indol, skatol, etc. The aromatic sulphates (*e.g.*, indican) are derived from the decomposition of proteid matter in the intestine and in the tissues. Barium chloride solution gives a precipitate insoluble in nitric acid with the inorganic sulphates, but not with the aromatic or ethereal sulphates.

The aromatic sulphates in the urine are increased in intestinal obstruction, peritonitis, etc., owing to the retention and increased putrefaction of the intestinal contents.

Cystin.

Cystin is possibly a normal constituent of the urine in minute quantity. In rare instances it gives rise to calculus, or to a sediment. The crystals are characteristic hexagonal plates. Cystinuria sometimes occurs in several members of a family, and may persist for years.

Bile.

Bile is present in the urine (*choluria*) in jaundice, and has been already considered in the account of icterus (p. 527).

Sugar, Diacetic Acid, Acetone, and β-Oxybutyric Acid as constituents of the urine have been considered in connection with diabetes.

Albuminuria.

Albuminuria is one of the most constant accompaniments of kidney disease, but each may be present without the other. The specimen of urine examined should as a rule be taken from the amount collected for the whole twenty-four hours, but it is often advisable to examine separately that passed on rising in the morning and that passed after meals or after exercise. Of the numerous tests for albumen (serum-albumen) in the urine, two are particularly good, and as a rule one of these is quite sufficient.

(1) The *heat test* is suitable for any urine, and is the best if the specimen is cloudy with urates. The first thing to do is to note the reaction. If the urine is not acid, it may be acidulated at once with acetic acid, or this may be done after the heat has been applied. A considerable quantity of the urine should be put into the test-tube so that the upper part may be boiled whilst the lower part of the tube is held between the fingers. If albumen is present, it is coagulated by boiling, and the urine is rendered cloudy or opaque. If the urine is alkaline or but faintly acid, a cloud may be produced by phosphates, but this at once disappears on the addition of a few drops of acetic acid.

Certain sources of fallacy ought to be borne in mind. (*a*) If the urine is alkaline, the albumen may not be precipitated (alkali-albumen). (*b*) If too much acid, and especially nitric acid, be added, the albumen may not be precipitated (acid-albumen). (*c*) Sometimes, in urine which is frankly acid, boiling yields no precipitate, and yet the subsequent addition of acetic acid gives a cloud. This is probably mucin in ordinary cases. The phenomenon, however, is sometimes well marked in urines which contain bile, and there is good ground for the view that in this case the cloudiness is due to bile acids which are liberated from the bile salts by the addition of acid.

If the urine is turbid with urates, it clears on heating. If turbid from phosphates, it may be cleared by acidulation or filtration. If turbid from decomposition, it may require repeated filtrations.

(2) *Heller's test* is performed with nitric acid in the cold. After the urine has been put into the test-tube, the tube is held nearly horizontally, and nitric acid is allowed to trickle gently down the side of the tube so as to form a layer at the bottom without mixing with the urine. Or the nitric acid may be put into the tube first, and the urine floated on the top of it. Where urine and acid are in contact, an opaque layer is produced if albumen is present. A change in colour does not mean albumen, but the production of a derived pigment.

Fallacies may be met with in this test also. (*a*) A cloud of urates may appear after the acid is added, but these are at some distance above the plane of contact, and, moreover, disappear on heating. (*b*) Cloudiness may be due to the presence of a resinous acid, *e.g.*, in a person taking copaiba ; but this is removed by heat, and the odour of the resin can be recognised in the urine. (*c*) If the nitric acid is simply poured into the urine, albumen may escape detection owing to the formation of acid albumen which is soluble. (*d*) If albumen is present in very small quantity, a period of seconds or even a few minutes may elapse before the haze appears. (*e*) If the urine is concentrated, *e.g.*, in pneumonia, a cake consisting of crystals of nitrate of urea may form at the plane of contact. The appearance, however, is quite different from that of coagulated albumen.

(3) *Johnson's picric acid test* is also employed. The urine must be acidulated if it is not acid to begin with. A saturated solution of picric acid is then floated on the surface of the urine, and if albumen is present, an opaque layer will form at the plane of contact. The test is quite reliable if the result is negative, but other substances besides albumen (urates, alkaloids, peptones) may be precipitated. These, however, dissolve on heating and reappear on cooling.

Other tests for albumen are not necessary when the heat and nitric acid tests are available, but one or two more may be mentioned.

(4) Solution of *potassium ferrocyanide* added to urine which is acidulated with acetic or citric acid produces a cloud if albumen is present.

(5) A saturated solution of *salicyl-sulphonic acid* may be used to distinguish albumoses and peptones from albumen. All of these substances, if present in an acid urine, give a precipitate with this test. If the turbidity is due to albumoses or peptones, it disappears on heating and returns on cooling. If it is due to serum-albumen, it does not disappear on heating.

Quantitative Tests for Albumen.—(1) The proportion of albumen in urine may be estimated in a rough way by half filling a test-tube with the urine, *boiling* it all through, acidulating it with acetic acid, and then allowing it to stand upright for twenty-four hours. The proportion of coagulum which settles at the bottom to the height of urine is then noted.

(2) *Esbach's apparatus* is a special form of test-tube, which is used with the following reagent, viz., picric acid, 35 grains, citric acid, 70 grains, water to 8 fluid ounces. The urine is filtered. If it is not distinctly acid, acetic acid is added drop by drop till the mixture changes litmus paper to a brick-red. If the specific gravity exceeds 1010, the urine should be diluted with one or more volumes of water to bring it below that density ; or if on the first trial the urine proves to contain more than 4 per 1,000 of albumen, it should be diluted. In either of these cases, the indication afterwards given by the deposit must be multiplied according to the degree of dilution.

With the urine thus prepared, the tube is filled to the line marked U. The reagent is then added up to the line marked R. The tube is thereupon closed with a plug and turned upside down several times so as to thoroughly mix without frothing the contents. The tube is allowed to rest for twenty-four hours in the upright position, and the amount of the deposit is then read off on the scale. The number indicates the parts of albumen per 1,000 of urine. If the urine was diluted, the necessary multiplication must of course be made.

(3) *Roberts's method* consists in diluting the urine with water until the haze caused by the addition of nitric acid becomes visible only after from thirty to forty-five seconds.

When this is the case, albumen is present in the proportion of 0·0034 per cent., or 0·0148 grain per fluid ounce. From the degree of dilution the richness in albumen of the undiluted urine can be estimated.

Significance of Albuminuria.—Urine which contains blood or pus gives the reaction for albumen; these substances may come from the kidney or from any other part of the urinary tract, or from an abscess which bursts into that tract, or, in the case of females, from the generative system.

For the physician, however, the most common cause of albuminuria is structural disease of the kidneys. The albumen is, to some extent, an inflammatory exudation, but in great measure it is the serum albumen circulating in the bloodvessels which escapes with the urine, because the damaged renal cells can no longer prevent it. In ordinary circumstances the escape of albumen takes place at the Malpighian tufts, but it is possible that the desquamation of epithelium from the tubules may also allow it to escape through the basement membrane.

Passive hyperæmia of the kidneys, as in heart disease, often causes albuminuria. The febrile state, however induced, is often associated with albuminuria, probably because the toxins in the blood damage the renal epithelium. The urine may be albuminous after epileptic seizures, and in connection with various other nervous disorders. Albuminuria may result from embolism of the renal artery.

Functional Albuminuria.

Albuminuria may occur without any discoverable lesion of the kidneys. One form which is common in adolescents is often discovered accidentally, for instance in the course of examination for life insurance. This is spoken of as *functional* or *physiological albuminuria* or the *albuminuria of adolescence*. It may be present at one part of the day and not at another, and hence is spoken of as *intermittent*, *remittent*, *paroxysmal*, *transient*, or *cyclical*. This kind of albuminuria has been ascribed to muscular effort, particular kinds of diet, dyspepsia, and cold bathing; but the most common variety,

in my experience, is *orthostatic* or *postural*. In this type the urine passed on waking in the morning is free from albumen, but that which is passed after the individual has been going about for a short time is albuminous. Gradually, as the day wears on, the albumen tends to disappear. If the individual lies in bed all day there is no albuminuria. The explanation is, apparently, that the renal circulation in these persons cannot accommodate itself promptly to changes in posture. Such persons are apt to be pale, lacking in robust vigour, or of a neurotic disposition, though no other evidence of disease can be detected. A schoolboy may faint when standing in church or at drill. Functional albuminuria may be present in more than one member of a family.

Diagnosis.—If the albumen is discovered accidentally, specimens of urine passed at different periods of the day ought to be separately examined, and some of them, most likely those passed immediately on rising, will be found free from albumen. Moreover, other signs of kidney disease will be absent. The heart is not enlarged, the pulse tension is likely to be low, and few or no tube casts will be detected in the urine. In the case of adolescents who, without definite signs of disease, suffer from pallor and languor, this variety of albuminuria should be looked for.

Prognosis.—This is good as regards survival and recovery from albuminuria. Functional albuminuria need not prevent an adolescent from being accepted for life insurance at ordinary rates.

Treatment.—It may be desirable to give a tonic mixture, containing a mineral acid ; and to this a little digitalis may be added if the condition depends upon posture, and the arterial tension is low. The subjects of this form of albuminuria should be allowed to join in all the games and exercises of their fellows, and should regard themselves as healthy individuals. Candidates who have to pass a medical examination may escape rejection in some cases by taking a mercurial purge on the preceding evening.

Albumosuria.

Albumoses are found in the urine in small quantity in various febrile and other diseases, where they are of no special importance. There is one rare disease, however, where albumosuria is of grave significance, viz., *myelopathic albumosuria* (*Kahler's disease*). The body excreted in this affection is known as Bence-Jones's albumose or Bence-Jones's proteid. Most of the cases have been in adult males, and in the great majority there have been multiple tumours of bone-marrow (myelomata). In some instances there is a history of severe injury to the bony skeleton. The disease usually proves fatal in less than three years.

The albumose is precipitated when cold nitric acid is added to the urine drop by drop ; it disappears on subsequent boiling, and it reappears on cooling.

Hæmaturia.

If blood is present in the urine in large quantity, the latter may be quite red. With smaller proportions of blood, the urine is brown or smoky. The blood tends to fall as a chocolate-coloured sediment. It may be recognised by the guaiac reaction, the microscope, and the spectroscope.

(1) To a small quantity of urine in a test-tube, tincture of guaiacum is added. The guaiac resin is precipitated by the water and renders the urine turbid. Ozonic ether (ethereal solution of hydrogen peroxide) is then added, and the mixture is shaken up. If hæmoglobin is present, a blue colour is produced, owing to oxidation of the resin. The blood takes oxygen from the ozonic ether and then gives it up to the resin. If the appearance of the urine suggests that the blood is scanty, the portion to be tested should be taken from the bottom of the glass, as the corpuscles tend to sink thither, carrying the hæmoglobin with them. A possible fallacy exists. With urine which contains iodine, *e.g.*, owing to the administration of potassium iodide, or the use of iodoform as a surgical dressing, a reaction may be obtained which suggests the presence of blood. The distinction is made by adding nitric acid ; this simply discharges the blue

colour if it is due to hæmoglobin, but replaces the blue by yellow or brown through the liberation of iodine if the latter is present. It used to be said that the presence of saliva in urine was another source of fallacy, but this is a mistake. Pure saliva gives no guaiac reaction.

(2) The microscope reveals the red corpuscles in the urinary sediment. These are never collected in rouleaux. They are ordinarily biconcave with a double outline, but may be shrunken or crenated. In dilute urine they tend to swell up, and may appear simply as faint rings. They may resemble spores, but the latter are generally oval and nucleated.

(3) The spectroscope reveals the absorption bands of one or other form of hæmoglobin ; viz., for oxyhæmoglobin, two dark bands, one being in the yellow and the other at the junction of yellow and green ; and for methæmoglobin, two bands in the situations just mentioned, with a third one in the red.

Urine containing blood gives the reactions for albumen.

Source of the Hæmorrhage.—Blood from the kidney is intimately mixed with the urine. If the blood comes from the bladder, it may appear at the end of micturition. If it comes from the urethra, it will exude between the acts of micturition. But in all cases it is important to be guided by the other data furnished by examination of the urine, and by the concomitant symptoms.

Etiology.—Hæmaturia may be due to a lesion of, or extending to, any part of the urinary tract. It may thus be due to trauma, tumour, parasites or calculus. It also occurs in acute inflammation of the kidneys, in turpentine and cantharides poisoning, and in the hæmorrhagic group of general diseases.

Hæmoglobinuria.

Hæmoglobinuria is a manifestation of hæmoglobinæmia, a condition in which the red corpuscles are dissolved in the blood-stream so that their hæmoglobin is set free. It has been considered in detail in connection with diseases of the blood (p. 363).

Pyuria.

Pyuria may result from suppuration in any part of the urinary system, or from an abscess opening into the urinary tract. In women it may be due to leucorrhœa. The source of the pus can often be recognised by associated symptoms and physical signs, but it is to be noted that if the urine is ammoniacal the pus is probably derived from the bladder. Even in these cases, however, the kidneys may ultimately become the seat of suppurative inflammation. A sediment of pus is dense and white or cream coloured, and the addition of liquor potassæ makes it gelatinous. The microscope reveals the pus corpuscles, which, on the addition of acetic acid, display their tripartite or bipartite nuclei. Urine containing pus gives the reactions for albumen.

Tube Casts and Epithelium.

Casts of the uriniferous tubules are a common feature in kidney disease, and are occasionally observed in small numbers in cases where it is almost certain that no renal disease exists. Tube casts vary in their characters according to the region of the tubules in which they are formed, according to the material of which they consist, and according to the form of kidney disease with which they are associated. Thus, most of them are straight, some are curved. They may be broad or narrow. The *hyaline* cast, which is the simplest and of least significance, is a cylinder of transparent hyaline matter with but little refracting power. *Waxy* (or *colloid*) casts are more highly refracting and often of large size. They sometimes give the reactions with iodine and methyl violet, but this is no certain evidence that amyloid degeneration exists in the kidneys. Other casts are *granular*, the granules being due to degeneration of epithelial cells, blood corpuscles, etc. *Epithelial* casts are cylinders made up of renal epithelium welded together, or moulded on a basis of hyaline matter. *Fatty* casts contain globules of oil, and are important as suggesting chronicity and therefore imperfect cure. *Blood* casts are made up of blood corpuscles. In rare cases *pus* casts are observed.

Hyaline and granular casts are observed in all kinds of Bright's disease. Epithelial and blood casts are specially related to tubular nephritis, whether acute or chronic.

Cylindroids are long, ribbon-like bodies, not unlike hyaline casts, but longitudinally striated. They may occur in health, and it is not certainly known whether they come from the kidneys.

Epithelium in the urine may come from any part of the urinary tract, and in females from the generative system. Thus, round cells with a single nucleus come from the uriniferous tubules, but may also be derived from a lower part of the tract. Cells of various shape, oval, tailed, etc. (*transitional* epithelium), and sometimes adhering to one another so as to form tessellated masses, may come from any part of the tract, from the pelvis of the kidney to the urethra. Very large flat cells from the vagina are often observed in the urine of females.

3. SUPPRESSION OF URINE (Anuria).

Suppression must be distinguished from retention of urine. In the former case no urine enters the bladder from the kidneys ; in the latter the urine reaches the bladder, but cannot escape from that viscus. Habitual or frequently recurring retention is apt to lead after a time to changes in the urinary system above the bladder, with the result that suppression may be brought about.

Suppression is of two kinds : (1) obstructive and (2) non-obstructive.

(1) *Obstructive* suppression may result from the blocking of one ureter by a calculus in a person in whom the opposite ureter has been already blocked, or in whom the opposite kidney is disorganised or absent. Or a cancer of the uterus may cause obstruction of both ureters near the bladder. Even with absolute suppression no distinctive symptoms set in for about a week (*latent uræmia*). There may be insomnia and some muscular enfeeblement, but the mental functions are sound, and the patient seems to have nothing particular wrong with him. During this week some urine is generally

passed, but in a most irregular manner as regards the times of micturition. Moreover, on account of the high pressure within the renal pelvis and uriniferous tubules, the urine which is secreted is pale, of low density and poor in urea ; it is generally free from albumen. Of the distinctive symptoms, muscular twitchings are the most important. Then follow contraction of the pupils, great weakness, dryness of the tongue, anorexia, slight delirium and death within two or three days. Convulsions, dropsy, and an ammoniacal odour of the breath and skin are quite exceptional.

(2) *Non-obstructive* suppression occurs in cholera, in acute nephritis, after the passage of a catheter, and after lesions of the abdomen. In acute nephritis the suppression is seldom complete for any length of time ; it is to be attributed to the changes in the renal cells. After the passage of a catheter, and in some abdominal cases, the suppression is apparently reflex, and due to changes in the renal blood-vessels (probably spasm). If the suppression is prolonged, acute uræmic symptoms develop. Anuria also occurs as a manifestation of hysteria, but it is commonly accompanied by vomiting of a fluid which contains urea, and it may be followed by polyuria ; it does not lead to uræmia.

Treatment.—In obstructive suppression the surgeon may occasionally be able to remove the obstruction ; the attempt should be made early. The treatment of non-obstructive suppression will be described in connection with nephritis.

4. URÆMIA.

This condition has been alluded to in connection with the subject of suppression of urine. It may be brought about by any condition which interferes with the secretion of urine (except, perhaps, hysteria). It is natural to suppose that the symptoms are due to retention in the system of excretory products which would be removed if the kidneys were healthy ; but while this is so, it is impossible at present to name any particular poison which can be held responsible for the symptoms, or to state exactly the mode in which these are brought about. It has also been suggested that

uræmia is due to perversion or loss of the internal secretion of the kidneys.

Acute Uræmia.—In this form the symptoms develop acutely, or even suddenly. Convulsions are common, and are usually epileptiform in type (*uræmic eclampsia*). They may be severe or slight as regards the amount of spasm. One may follow on another almost immediately (*status epilepticus*), or there may be an intervening period of coma. Coma itself is an important symptom. With numerous or severe convulsions the temperature may rise to 104° or thereby. Coma and convulsions occur both at the onset of acute nephritis and as terminal phenomena in the chronic disease. Another symptom is blindness without ophthalmoscopic change (*amaurosis*). Delirium, insanity and hemiplegia (not due to any gross cerebral lesion) may also occur. Death may take place in the convulsive or in the comatose period. Recovery, however, is common, since the attack of nephritis often subsides.

Chronic Uræmia.—Here the symptoms are less urgent, though more varied, but they may at any time become acute. Headache is common and often severe, especially in the morning. Vomiting, diarrhœa, anorexia, a rapid pulse, insomnia, muscular weakness, and erythematous and other affections of the skin also occur. Dyspnœa may be troublesome, and the continuous variety is occasionally so severe as to cause death by exhaustion. In chronic uræmia the temperature is usually subnormal (unless inflammatory complications are present), and a very characteristic mode of termination is a gradual fall of temperature accompanied by a gradually increasing drowsiness, going on day after day until the latter ends in coma, and after a time in death, with or without a few convulsions.

In Bright's disease the knee-jerks are frequently altered from the normal. They may be diminished, absent, or increased.

5. DROPSY IN KIDNEY DISEASE.

The characteristic dropsy of kidney disease differs from that of cardiac disease or weakness in not showing a preference for the most dependent parts. It is true that dropsy of the *cardiac* type is met with in renal disease, but this is due to secondary or coincident disturbance of the heart. The distinctively *renal* dropsy includes both anasarca or general œdema—involving more or less all parts of the surface, but especially parts where the cellular tissues are loose (*e.g.*, the eyelids)—and dropsy of the serous cavities. Once the anasarca has become considerable, the fluid naturally tends to gravitate to the most dependent parts—*e.g.*, the side on which the patient habitually rests. To explain renal dropsy we must suppose that the permeability of the vessels is increased either by toxic substances which are retained in the blood owing to the loss of the renal function, or by the toxic substances which caused the nephritis. Renal dropsy is specially associated with tubular nephritis, whether acute or chronic.

6. CARDIO-VASCULAR CHANGES IN KIDNEY DISEASE.

Changes in the kidneys and circulatory organs may be associated with one another in three ways. (1) The changes in both may be due to a common cause. For instance, the poison of diphtheria may cause nephritis on the one hand, and degeneration of the cardiac muscle with dilatation of the ventricle on the other. (2) Disease in the heart may give rise to changes in the kidneys. For instance, rheumatic endocarditis may give rise to embolism in the kidney,[1] and in the same way ulcerative endocarditis may lead to the formation of abscesses in the kidneys. (3) Disease of the kidneys is often the cause of changes in the heart and blood-vessels, and especially of hypertrophy of the left ventricle.

[1] A condition long ago described by P. Rayer (*Maladies des Reins*, 1839-41, ii. 73) as 'néphrite rhumatismale,' and not to be confused with the acute nephritis which in rare instances shows itself as a complication of rheumatic fever.

This, of course, is chiefly witnessed in chronic disease. This subject, however, and in particular the relationship between changes in the kidneys and changes in the arteries, will be considered in connection with chronic Bright's disease.

The hypertrophy of the left ventricle is due to increase of arterial tension. As to the immediate cause of the increased tension, different opinions have been held in the past, but there is very good ground for the belief that it results from the morbid state of the blood, contaminated as it is by excretory products which the kidneys have failed to eliminate. This abnormal blood, according to Johnson's stop-cock theory, irritates the tissues and causes reflex contraction of the arterioles, thus tending to protect the tissues by shutting off some of the impure blood. Or the obstruction may be primarily, as Broadbent thinks, in the capillaries. The high tension present in the early stage of anæmia suggests that abnormal blood does not glide through the capillaries so easily as normal blood. But whether any of these agencies is the actual one or not, the fact remains that the increased resistance to the arterial outflow leads to increased arterial tension and hypertrophy of the left ventricle ; and as the increase of tension and the cardiac hypertrophy have the effect of driving an increased quantity of blood through the kidneys, and thus giving these organs a better opportunity of separating the excretory products from the blood, the cardio-vascular changes may be regarded as thus far compensatory in their nature. Unfortunately, however, in the long-run, the abnormal strain tells upon the vessels and heart, and degeneration ensues.

7. DISTURBANCES OF THE CIRCULATION IN THE KIDNEYS.

Active Hyperæmia.

Active hyperæmia results from the action of irritants such as cantharides, turpentine, and the toxins of scarlatina and other fevers, but it is doubtful whether such congestion should be distinguished from inflammation. Occasionally,

however, intense congestion of reflex origin would appear to be the explanation of the *urethral shock* which may follow the passage of a catheter. The kidneys are large, their vessels are engorged, and their capsules strip off easily. The urine is scanty and albuminous, and often contains blood. There is no dropsy.

Passive Hyperæmia.

This is common as a part of the general venous congestion which results from failure of the heart in valvular disease, emphysema, etc. It is also produced by pressure on the renal veins or inferior vena cava by tumours, etc., in the abdomen. The kidneys are large, firm and deep red, and their capsules strip off easily—at least in the early stages. The urine is scanty and concentrated. It contains albumen, and sometimes blood and hyaline tube casts. As a rule, this condition does not give rise to symptoms. The treatment is that of its cause.

Embolism.

Embolism is common in heart disease, and especially in mitral stenosis, where a thrombus often forms in the left auricle. Or a piece of thrombus may come from the left ventricle, or from the interior of an aneurysm; vegetations may become detached from the valves of the heart in endocarditis; or, again, calcareous matter may be removed from an atheromatous patch in the aorta.

Embolism of the kidney leads to necrosis of tissue. The usual clinical evidence of embolism is the sudden onset of albuminuria of a temporary kind in a patient suffering from some disease likely to be associated with embolism. Sometimes the albuminuria is accompanied by hæmaturia and by pain in the loin.

If the embolus is septic, as in ulcerative endocarditis and pyæmia, an abscess may be produced. Such lesions are commonly multiple and small. Septic embolism may be accompanied by a rigor and followed after some days by pyuria.

8. BRIGHT'S DISEASE.[1]

Great diversity of opinion exists as to the clinical and anatomical types of disease which ought to be included under this designation. In the present state of our knowledge, however, the most convenient classification is one that has long been in general use. By this arrangement, three varieties of Bright's disease are recognised : (1) In the first form, the onset is acute, and the course acute or subacute. While all the tissue elements suffer, the tubules suffer most as a rule (acute tubular nephritis), but sometimes the vessels of the glomeruli are most involved (glomerular nephritis). (2) In the second form the onset is usually acute, though sometimes gradual, and the course is chronic. Here, again, the tubules chiefly suffer (chronic tubular nephritis). (3) In the third type, the onset is insidious and the course is chronic ; here the interstitial tissue is principally involved (chronic interstitial nephritis). (4) In connection with these three types amyloid degeneration of the kidney (which is not a form of Bright's disease) may be mentioned here ; first, because it is associated, like Bright's disease, with albuminuria and dropsy ; secondly, because it is apt to be accompanied by inflammatory changes in the kidneys ; and, thirdly, because, like the second and third types of Bright's disease, it may at any period of its course be complicated by an acute or subacute attack resembling the first variety. Indeed, a considerable proportion of cases which, when admitted to hospital, appear to be examples of acute Bright's disease are found on investigation to be acute or subacute exacerbations of a chronic affection.

Saundby classifies nephritis according to its etiology, and distinguishes three types : infective, toxic and obstructive. The *infective* type includes all cases of acute or chronic nephritis, which occur as a result of acute or chronic infective diseases. The *toxic* group includes cases due to lithæmia or the uric acid diathesis, and corresponds especi-

[1] So named after Richard Bright, who, in his ' Reports of Medical Cases,' published in 1827, called attention to the connection of dropsy and albuminuria with changes in the kidneys.

ally to the contracted, red, granular kidney, but the kidney may be large and fatty owing to intercurrent acute or subacute attacks. It also includes the acute nephritis of acute gout ; that which results from poisoning by animal, vegetable and mineral poisons ; and that which results from chill. In the toxic group, the inflammation is attributable to irritation of the renal tissues by the abnormal substances which they eliminate. The *obstructive* type includes cases due to obstruction to the urinary outflow, *e.g.*, by enlargement of the prostate or stricture of the urethra in males, or by the pressure of an enlarged uterus or a pelvic new growth in females. Obstructive nephritis is practically synonymous with *consecutive* nephritis (the nephritis being consecutive to disease lower down the urinary tract), but both expressions include certain forms of suppuration of the kidney, whereas suppurative nephritis is best kept apart from Bright's disease altogether.

In each of Saundby's three types, the urinary and other symptoms may point to either acute or chronic nephritis, and the anatomical changes may be those of acute nephritis, chronic fatty kidney, or contracting kidney.

Acute Nephritis (Acute Bright's Disease).

Etiology.—Exposure to cold and wet is a common cause, especially in adults, and is more potent if the individual is under the influence of alcohol. Scarlet fever is a very common cause, and is naturally most influential in childhood. The symptoms usually appear in convalescence, and cannot be accounted for by exposure. Other fevers are much less frequent causes. Pregnancy is a well-known cause. Certain poisons which have been mentioned as causing active hyperæmia (*e.g.*, cantharides and turpentine) are probably capable of exciting actual inflammation.

Morbid Anatomy.—In some cases (*e.g.*, glomerular nephritis), the kidneys may appear almost healthy to the naked eye. In other cases, they are swollen and dark, and on section are found to be engorged with blood. In other cases, while the pyramids are dark red, the swollen cortex is grayish-red and the surface is pale. In any case the capsule is easily

removed. The microscope shows that the inflammation is diffuse and involves all the tissues, although in different cases different tissue-elements suffer most. In the tubules, there is cloudy swelling of the epithelium, which tends to desquamate so that the tubules may be distended with cells. After the early stages the epithelium may be very fatty. In the tubules there are not only epithelial cells, but also tube casts and often red blood corpuscles or brown granular débris. The Malpighian tufts may be seriously compressed by swelling of the glomerular epithelium, or by hæmorrhage or round-cell infiltration into Bowman's capsule, so that the secretion of urine may be arrested. The interstitial tissue is the seat of round-cell infiltration and sometimes of hæmorrhage.

Parenchymatous, *desquamative*, *tubal*, *tubular*, or *diffuse* nephritis is the type ordinarily met with as the result of exposure. *Glomerular* nephritis (*glomerulo-nephritis*) is the variety specially seen in scarlet fever.

Symptoms.—These vary a good deal. The patient may complain of nothing except the swelling, the onset of which is often abrupt (*acute renal dropsy*). There may be some chilliness with slight elevation of temperature at the outset, but there is often no fever. Pain in the back is not common. There may be vomiting or convulsions at the commencement. The swelling generally begins in the eyelids and about the ankles, and may soon involve the general surface. The dropsy may involve the lungs and serous cavities. Pallor is a marked and early feature of the disease.

The urine is passed with undue frequency. It is scanty, altered in colour, and of high specific gravity. The colour is usually smoky, brown or red, though occasionally normal. Abundant albumen is present, together with tube casts, and often blood. The tube casts are epithelial, blood, granular, hyaline and perhaps fatty. Desquamated renal cells are also present. The urea excretion is reduced.

In severe cases blindness (*amaurosis*) may be present. The loss of sight is complete, and comes on suddenly; it passes off again quickly if the state of the blood is rectified. As the pupils respond to light in some cases and not in

others, it is probable that the uræmic poison acts sometimes on the cortical visual centres, and sometimes on the retinal nerve elements with or without the centres. The condition is not related to any ophthalmoscopic change. In this form of Bright's disease, the fundi are generally normal.

The tension of the pulse is raised (see sphygmogram, Fig. 19, p. 265). Bronchial catarrh is generally present.

Diagnosis.—This is usually simple. The ordinary albuminuria of acute fevers is not to be regarded as due to Bright's disease. Myxœdema is readily distinguished from Bright's disease by the examination of the urine. When a case comes under observation presenting symptoms of acute nephritis, it is important to search for any evidence of pre-existing chronic disease (hypertrophied or degenerated arteries, hypertrophied heart, albuminuric retinitis, etc.), as this will greatly modify the prognosis.

Prognosis.—A large proportion of cases recover. The symptoms subside in the course of days or weeks, the urine becomes more abundant than normally, and its abnormal ingredients gradually diminish and disappear. The albuminuria may persist for months, though it occasionally ceases within a few weeks. Other cases die in the acute stage from uræmia, or occasionally from serous effusion or inflammatory complications. Secondary inflammations, however, are not nearly so common in acute as in chronic Bright's disease. Other cases merge into a chronic condition.

Treatment.—The patient should rest in bed between blankets. Milk diluted with water, barley-water, or soda-water, is a good food, and it is a common practice to restrict the patient to diluted milk during the acute stage; the milk being regarded as a complete and non-irritating food, the lactose as a diuretic, and the water as a bland agent which flushes out the kidneys, removing desquamated epithelium, blood corpuscles and tube casts, which tend to obstruct the tubules. But there is reason to doubt the wisdom of introducing unusually large quantities of liquid into the system as long as the kidneys are unable to excrete even the normal amount; and it is therefore right to restrict

the liquids to about 3 pints daily, and to supplement the dietary by farinaceous foods, until a distinct increase in the quantity of the urine shows that the kidneys are beginning to overtake the arrears of their work. If in the early stage the urine is extremely scanty and the dropsy is decidedly threatening, the total dietary should be restricted to about a pint of milk, and ice may be given to relieve the thirst. After the acute stage is past, liquids as well as farinaceous foods may be given quite freely. Diluted milk, alkaline waters, weak tea, and imperial drink (cream of tartar solution, sweetened with sugar and flavoured with lemon-juice) may be recommended as drinks; while porridge, rice, gruel, cornflour, and bread are available as foods in addition to the milk. Still later, fish may be given.

At the outset the bowels should be freely purged by compound jalap powder (1 drachm for an adult), and constipation must be guarded against all through the attack. Solution of ammonium acetate may be given at first as a diaphoretic. It is an old-established practice to give digitalis and other diuretics in this disease, but their usefulness in the stage of early oliguria may be questioned.

If the symptoms are threatening, the loins may be cupped at first and then poulticed; in young vigorous subjects, wet cups may be employed. A hot wet pack conjoined with hot drinks is a valuable means of promoting perspiration. A hot-air bath is sometimes used. Pilocarpine ($\frac{1}{8}$ to $\frac{1}{4}$ grain for an adult, $\frac{1}{20}$ to $\frac{1}{12}$ grain for a child) may be employed with caution if the pulse is good, and if there is no evidence of moisture in the bronchial passages. If dropsy is severe, Southey's tubes should be used for the superficial tissues, and the aspirator for the serous cavities. Another useful measure in dropsical cases is to withdraw common salt from the dietary. This is sometimes followed by most encouraging results, as shown by diminution of the body-weight, the dropsy, and the albuminuria; increase in the quantity of urine and urea; and relief of uræmic symptoms. Convulsions may be controlled by chloroform, while potassium bromide and chloral are administered by the rectum, and cups and poultices are applied over the kidneys. Morphine

is frequently given for uræmic convulsions, and may act well, but it is open to the objection that it may be difficult to distinguish between morphine poisoning and uræmic coma. For obstinate vomiting, ice, bismuth and dilute hydrocyanic acid may be given by the mouth, and a mustard poultice may be applied to the epigastrium.

After the acute stage is passed, some chalybeate should be given for the anæmia, but the state of the urine should be carefully watched lest the iron irritate the kidneys. The patient must always be well protected by flannel clothing.

Chronic Parenchymatous Nephritis

(Chronic Desquamative, Tubal, Tubular, or Diffuse Nephritis).

Etiology.—This is the chronic form of nephritis which follows an acute attack, whether the latter be due to exposure, to scarlet fever or to pregnancy. Occasionally, however, the onset is insidious, and various agencies such as alcohol, habitual exposure and infectious diseases have been suggested as causes. This form of the disease occurs under the same circumstances as, and frequently in combination with, amyloid degeneration of the kidneys. It is most common in early and middle adult life.

Morbid Anatomy.—The kidneys are usually enlarged. The surface is smooth and pale, with conspicuous stellate veins, and the capsule is easily removed. The cortex is swollen and pale, contrasting in colour with the red pyramids. The tubules are distended with swollen, fatty and desquamating epithelium. When the large white kidney contains much fat, it constitutes one form of *fatty kidney*. Casts are also seen, Bowman's capsules are thickened, and the interstitial tissue is infiltrated with round cells. This is the *large white kidney*. In some cases there are hæmorrhagic areas in the cortex, and the condition is then termed *chronic hæmorrhagic nephritis*.

The *small white kidney* (*pale granular kidney*) is generally regarded as a more advanced stage of the disease than the large white kidney, although, on the one hand, the large

white kidney may not become small, even though the patient survive for several years ; and, on the other hand, it is believed by some that the small white kidney may never have had a stage of enlargement. The kidneys are nearly normal as regards size, somewhat pale, and rough on the surface, with adherent capsule. The cortex may be thinner than normally. There is overgrowth of interstitial tissue, with atrophy or sclerosis of Malpighian tufts, thickening of arteries, and degeneration of epithelium. The changes, in short, are intermediate between those of the large white and the small red kidney.

The left ventricle is usually hypertrophied, and the walls of the arteries may be thickened.

Symptoms.—These are often a continuation or a relapse of the symptoms of acute nephritis. In the primary cases, there may be a general failure of health, with digestive disturbances, before swelling is noticed about the ankles and eyelids. In the fully developed disease, dropsy and pallor are almost constant phenomena. The whole body may be bloated with œdema, and the serous cavities are often the seat of dropsical effusion. The urine contains a large amount of albumen and abundant tube casts (including epithelial, fatty, granular, and hyaline). In many cases, blood is present (*chronic hæmorrhagic nephritis*). The quantity and specific gravity vary in different cases, and may be either above or below the normal. The excretion of urea is reduced. The various symptoms of uræmia as previously described are common, and secondary inflammations of the lungs and serous membranes are also common. The pulse tension is usually elevated, and the left ventricle tends to become hypertrophied, but these and other cardio-vascular changes are not so striking as in chronic interstitial nephritis. Various inflammatory affections of the skin are met with. Retinal changes are common (*albuminuric retinitis*), and include white spots or patches due to degeneration and exudation ; hæmorrhages ; optic neuritis ; and a general haziness over the retina due to inflammation of that membrane. The retinal changes are specially apt to involve the region around the macula. If they are slight, they may not

interfere with vision, and in any case the eyesight is rarely altogether lost.

Diagnosis.—Chronic tubal nephritis is distinguished from the *interstitial* form by the history, by the persistent dropsy, and by the characters of the urine, which contains abundant albumen and casts, and often blood. In pure *amyloid* disease there is no blood in the urine ; there may be amyloid disease elsewhere, and there may be an obvious cause for the degeneration (chronic suppuration, syphilis, etc.).

Prognosis.—Occasionally recovery takes place after the disease has continued for a year or more, but as a rule the outlook is very serious. Many cases die within six months, the fatal termination being due to uræmia, secondary inflammation or dropsy. On the other hand, I have known a patient return to work after being absent from it for two years or more, and survive for another year. Retinal changes are seldom survived for two years, and many of the cases die within a few months after these are detected. Defects of vision may pass away, however, and the ophthalmoscopic changes may disappear completely many months before death.

Treatment.—When the patient first comes under observation, especially if there is considerable dropsy, it is well to keep him in bed for a short time ; not so much for the purpose of observation, as to give the kidneys the best chance of recovering from any exacerbation of the disease which may be present. Apart from this, however, and apart from the need of treating important symptoms, such as hydrothorax or extreme dropsy of the legs, there is nothing to be gained by confining the patient to bed or even to the house. He must, however, be well clad with flannels so as to avoid the dangers associated with chilling. He should be allowed considerable latitude in diet, and butcher's meat may be permitted once a day at least. A plain, varied fare which keeps the weight nearly constant is the one to be recommended. Alcohol and strong condiments should be forbidden. There should be strict moderation both in eating and in drinking, but no special restriction need be placed upon the amount of liquid taken, since experience shows,

as Von Noorden points out, that even in the œdema of this form of nephritis, dehydration of the organism is futile as a mode of treatment.

If the patient is able to travel, he might with advantage winter in a warm dry climate. The activity of the skin and bowels must be kept up. Iron should be given for anæmia, provided that it suits the patient. The effervescing citrate of caffeine is the best remedy for headache. Further details of the treatment are in the main similar to those recommended for acute nephritis.

Some interest was recently taken in the suggestion of Edebohls that decapsulation of the kidney should be performed for chronic nephritis, his proposal being based on the improvement which followed fixation of a movable kidney by the removal of its capsule. He thought that the additional blood which the organ would receive as a result of this operation would improve its nutrition. The results yielded by this operation in many cases where it has been performed do not justify its general adoption. Opotherapy[1] has also been employed, the aim being to administer to the patient the internal secretion of the kidneys which is lacking in his case: *e.g.*, by giving glycerin extract of kidney; by giving the juice obtained by macerating finely-minced fresh pork kidneys in salt water; or by injecting serum drawn from the renal vein of a goat.

Chronic Interstitial Nephritis

(Atrophic Nephritis. Contracted, Gouty, Granular, or Small Red Kidney. Fibrosis, Cirrhosis, or Sclerosis of the Kidney).

Etiology.—This disease occurs chiefly in the second half of life, and is more common in men than in women. Gout, chronic lead-poisoning, alcoholism, habitual overeating, repeated exposure, mental strain and heredity are among the causes, but very often no cause can be recognised. Good authorities believe that granular kidney occurs secondarily to arterio-sclerosis, and according to Osler this is by far the most common form in the United States.

[1] ὀπός, juice.

Morbid Anatomy.—The kidneys are much reduced in size, and the capsules are firmly adherent, so that when they are removed little pieces of kidney tissue are torn off with them. The surface is rough, being covered with the 'granulations of Bright.' These are relatively pale, and correspond to parts where tubules still exist, whereas the intervening depressions are reddish, and correspond to a more advanced condition of disease. The general surface appears red. Section shows that the cortex is greatly thinned. Cysts may be seen, and the walls of the bloodvessels are thickened.

Microscopical examination shows great increase in the fibrous tissue. If the changes are not very old, round-cell infiltration may still be discoverable. Many Malpighian bodies are sclerosed, the capsules being thickened and the tufts converted into a homogeneous glancing fibrous tissue. The branches of the renal arteries are thickened in all their coats, and the increase of the intima may almost block the small vessels (*endarteritis obliterans*). The tubules are extensively atrophied or destroyed, and these changes, together with the contraction of the fibrous tissue, cause the glomeruli to be crowded together in an irregular manner. Where the tubules are not yet destroyed, they may be distended by epithelium ; and the latter may be fatty, atrophied or detached. Cysts are produced by obstruction of the tubules.

The left ventricle of the heart is hypertrophied, and the arteries of the body generally are hypertrophied and often degenerated.

The *arterio-sclerotic kidney* is not much reduced in size, and the cortical atrophy occurs in patches.

Pathology.—Great differences of opinion have existed with regard to granular kidney. The best working hypothesis is that the lesion is a chronic diffuse inflammation of the kidneys, involving specially the fibrous tissue and leading to compression and destruction of the secreting structures, so that the excretory capacity of the organs is gradually diminished. There is reason to believe that impure blood passes through the capillaries and arteries of the body less easily than pure blood, and to overcome this difficulty in Bright's

disease, the left ventricle undergoes hypertrophy. The result is increased blood tension, and to withstand the increased strain, the arterial walls also undergo hypertrophy.[1] Great prolongation of the strain leads to arterial degeneration.

Gull and Sutton, however, in 1872, advanced the theory that the primary lesion was not in the kidneys, but in the bloodvessels of the body generally, including those of the kidneys (*arterio-capillary fibrosis*). The secreting tissue of the kidneys suffered secondarily to the vascular lesion in the kidneys (*arterio-sclerotic kidney*). The cardiac hypertrophy was attributed to the changes in the vessels.

According to a third theory, which is extensively held at the present time, the primary renal lesion is a degeneration of the secreting structures in the glomeruli and tubules, and the interstitial changes are secondary.

Symptoms.—The disease may exist for an indefinite time without causing any symptoms. The first evidence of its existence may be cerebral hæmorrhage, cardiac failure or sudden uræmia. But usually less urgent symptoms set in in a gradual manner. There may be a general failure of health, with loss of flesh, strength and appetite without obvious cause ; frequent headaches, occasional vomiting, and shortness of breath. The patient may have to rise several times at night to micturate. The urine is unduly abundant and of low specific gravity. It usually contains a small quantity of albumen, though this may be absent from time to time, and a few hyaline and granular casts may be discovered. In exceptional cases there may be transient hæmaturia. Dropsy is commonly absent till a late stage, or throughout, and when present is usually slight and of the cardiac type (unless subacute nephritis complicates the chronic disease). By the time the patient comes under observation the left ventricle is likely to be hypertrophied. The pulse tension is raised, the arteries are hypertrophied and perhaps degenerated, and the aortic second sound is accentuated. Changes in the retina are common, and include

[1] The hypertrophy of the muscular coat of the minute arteries in the kidneys and other parts of the body in Bright's disease was pointed out by George Johnson in 1852.

hæmorrhages, slight neuritis, and retinitis ; but the most characteristic of all in this disease are whitish streaks of degeneration radiating from the macular region. Failure of sight is sometimes the first symptom complained of by the patient. Eczema is a common complication, and diarrhœa, epistaxis, and purpura may also be met with. Bronchitis, pleurisy, pneumonia, and pericarditis are other complications which may occur. The knee-jerks may be altered in this as in the parenchymatous type of Bright's disease.

In *arterio-sclerotic* cases the urine, though albuminous, may be practically normal in quantity, specific gravity, and colour.

Diagnosis.—The most important signs of this disease are the albuminuria, scanty in amount and associated with polyuria ; the increased arterial tension ; the arterial thickening ; the hypertrophied heart ; the retinal changes ; the age of the patient ; and the insidious onset. The case may, however, first come under notice owing to the supervention of an acute or subacute attack. If it is first seen in the stage of cardiac failure, it will be necessary to consider the history carefully, in order to distinguish the condition from one of heart disease with passive hyperæmia of the kidneys.

Prognosis.—The disease is incurable, but with due care a patient may hope for many years of life with fitness for duty. Many cases die from cerebral hæmorrhage, owing to the degenerated arteries at length yielding under the abnormal blood-pressure. Another group die from failure of the heart under the prolonged excessive strain ; the left ventricle becomes dilated, and relative incompetence of the mitral valve ensues, with engorgement of the pulmonary vessels, dropsy, and the usual train of cardiac symptoms. Or, again, uræmic complications (vomiting, diarrhœa, coma) or secondary inflammations may be the cause of death.

Treatment.—Any recognisable cause should be removed. The patient should avoid worry and severe effort. The bowels must be regulated and the skin kept active. An occasional Turkish bath or hot-air bath may be useful. Only small quantities of butcher's meat should be taken, and alcohol should be avoided altogether. While in acute nephritis the kidneys must be spared, in cirrhosis of the

kidney the circulatory organs must be specially considered. Accordingly, the patient must observe strict moderation in food and drink, and it is to be noted, as the result of careful investigations by Von Noorden and others, that such moderation in drink involves no risk that the excretion of urinary solids will be reduced. The body should be well clad, and if possible the winter should be spent in a mild climate.

Iron is indicated if marked anæmia is present. If there is trouble from the persistent high tension, nitroglycerin may be given regularly, and a saline purge may be employed from time to time.

If uræmic symptoms threaten, the hot pack may be used, together with various other measures recommended for the treatment of acute nephritis. If there is no auscultatory evidence of moisture in the respiratory passages, and if the pulse is of fair strength, pilocarpine ($\frac{1}{8}$ to $\frac{1}{4}$ grain hypodermically for an adult) and hot drinks, with a dose of sal volatile, may be administered just before the patient is put into the pack.

For a failing heart strychnine is indicated, and in spite of the risks connected with degenerated arteries and perhaps a degenerated heart, it may be necessary to employ digitalis. Headache should be treated by a dose of the effervescing citrate of caffeine each morning. For restlessness, insomnia, delirium, and dyspnœa, morphine should be tried. Edebohls's operation of decapsulation for chronic Bright's disease has been referred to in connection with chronic parenchymatous nephritis.

9. AMYLOID DEGENERATION OF THE KIDNEYS

(Waxy or Lardaceous Disease).

Etiology.—Waxy kidney is met with in connection with syphilis and suppurative tubercular lesions, and occasionally in some other conditions. It is sometimes associated with chronic parenchymatous nephritis, the two being attributable to the same cause. The kidneys are seldom the only seat of amyloid change.

Morbid Anatomy.—The kidneys are usually enlarged and pale, and the capsule is easily removed. The cortex is thick and pale, contrasting with the red pyramidal region. The tissue has a translucent, bacon-like appearance. The Malpighian tufts and arteriæ rectæ suffer early ; they are stained brown when a solution of iodine is applied to the cut surface. The other arteries and the basement membrane of the tubules may suffer later. The epithelium becomes fatty. Interstitial change is often present.

The heart is not hypertrophied unless there is coincident Bright's disease.

Symptoms.—Albuminuria, dropsy of renal type, and anæmia are the principal evidences of amyloid kidney, especially when supervening in a case where there is or has been some disease which might account for amyloid degeneration. There may, moreover, be signs of waxy disease elsewhere (splenic enlargement, hepatic enlargement, or diarrhœa).

The urine is at first increased in quantity, pale and of low specific gravity, with but a small proportion of albumen. Later on it tends to become scanty, more dense and highly albuminous. Tube casts are few and mainly hyaline, unless nephritis is also present.

Cardio-vascular changes and uræmic phenomena are seldom observed if the renal change is purely amyloid.

Prognosis.—This is usually grave, especially if dropsy is present. If, however, the cause can be removed (*e.g.*, chronic suppuration connected with diseased bone), recovery may take place.

Treatment.—The cause must be removed if this is practicable. Good food, fresh air, cod-liver oil, chalybeates and other tonics are indicated.

10. HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calices of the kidney, resulting from obstruction lower down in the urinary tract and accumulation of non-purulent fluid.

Etiology.—The obstruction may be congenital, and is then usually in the urethra. Obstruction of the ureter is a

common cause. This may be due to a calculus ; or to a kink in the ureter, caused, for instance, by that canal arising from the upper part of the pelvis instead of from the lower, so that the pelvis becomes distended with urine and drags upon the ureter ; or to pressure from without, as by an abnormal renal artery or by a cancer of the uterus.

No great pressure is required to cause obstruction. The dilatation is much greater if the obstruction is incomplete or intermittent than if it is complete and persistent. In the latter case the pressure in the renal vessels is soon neutralised by the pressure in the pelvis and tubules, and the secretion is arrested ; whereas a temporary withdrawal of pressure allows of great hyperæmia and abundant secretion.

Morbid Anatomy.—The pelvis and calices are dilated, and there is atrophy of the renal tissue, which, beginning with the papillæ, spreads outwards in the substance of the kidney. The latter is at length converted into a cyst, which is occasionally so large as to constitute a distinct abdominal tumour. The ureter above the obstruction may be so dilated as to resemble small intestine. Hydronephrosis may be unilateral or bilateral, according as the obstruction affects one or both ureters ; but unilateral hydronephrosis may result from prostatic enlargement if the hypertrophied bladder happens to compress one ureter at its lower end. On the other hand, though one calculus blocks only one ureter, a similar condition is not unlikely to supervene on the other side.

The contents of the dilated pelvis resemble a very watery urine. In unilateral cases there may be compensatory hypertrophy of the opposite kidney. The left ventricle of the heart may be enlarged.

Symptoms.—The slighter degrees of unilateral, or even of bilateral, hydronephrosis may give rise to no symptoms. If the condition is well marked, a tumour develops in the flank, and becomes recognisable by palpation in front, or by bimanual palpation, as well as by percussion. The colon lies in front of this tumour, but may be so flattened as not to give a distinctly tympanitic note.

In some cases—*e.g.*, where the ureter rises from an unusual part of the pelvis—the hydronephrosis is *intermittent*. At

times the obstruction is removed, possibly on lying down in bed, with the result that the patient passes an immense quantity of urine and the tumour disappears for the time being.

In unilateral cases the urine is not much altered, as the opposite kidney undergoes compensatory hypertrophy. In bilateral cases there may be diminution in the quantity of urine.

Diagnosis.—In *pyonephrosis* there may be pus in the urine. In this condition and in *perinephric abscess* there are constitutional and local evidences of suppuration. An *ovarian tumour* comes more to the front of the abdomen, and is not crossed by the ascending or descending colon. Vaginal examination and the history of the case may also give important information. Intermittent hydronephrosis is recognised by its distinctive features. Sometimes it is desirable to use the aspirator for diagnostic purposes. In all cases the history and the existence of a possible cause of hydronephrosis must be taken into consideration.

Prognosis.—This depends much on the cause. Unilateral hydronephrosis is not necessarily dangerous, but there is the risk of a calculus blocking the opposite ureter. Bilateral hydronephrosis tends to cause death by uræmia. Cases due to uterine cancer are often bilateral, and death may then result either from the growth or from uræmia.

Treatment.—Unless there is distress, the condition may be left alone. Or the fluid may be removed by aspiration at a point midway between the lower costal margin and the ilium. This operation may be practised repeatedly. If this fails, the sac may be opened and drained. Possibly the obstruction may then be found and removed. If these measures do not succeed, and if the opposite kidney is sound, the sac may be removed altogether.

11. CALCULUS IN THE KIDNEY

(Nephrolithiasis).

Etiology.—In the *kidney substance* (1) salts of uric acid (mostly ammonium urate) are found in about half the children who die in the first few weeks after birth. These are deposited chiefly in the tubules near the apices of the pyramids, and appear as reddish streaks. (2) Crystalline masses of sodium urate are found in gout in the thickened fibrous tissue of the gouty kidney. (3) Lime salts are sometimes found in the straight tubules of the pyramids in the form of white streaks.

In the *renal pelvis*, calculi are common. They may originate there in consequence of chronic pyelitis, or they may have originated in the tubules and been washed into the pelvis. In the latter situation, they may be found varying in numbers from one up to hundreds, and in size from minute grains of sand or gravel to a calculus fully occupying the dilated pelvis and calices.

The most important stones are the uric acid calculus (reddish, hard, smooth, small or large, single or multiple), and the oxalate of calcium calculus (of brownish colour, very hard, and often rough like a mulberry). Other stones consist of urates, triple phosphate, calcium phosphate, calcium carbonate, cystin, xanthin, etc.

There are probably two factors which determine the formation of a renal calculus. (1) There is first a change in the urine. So far as the uric acid stone is concerned, this is favoured not so much by increase in the quantity of uric acid excreted as by diminution of the power of the urine to hold it in solution. High acidity and low density of the urine favour the deposition of uric acid. The deposition of calcium oxalate will naturally be favoured by the constant use of articles of food which increase the amount of that substance in the urine. (2) In the second place, there is probably often some local change in the pelvis which by giving rise to excess of mucous secretion, or to hæmorrhage, may furnish a nucleus upon which a calculus may grow.

Renal calculus is particularly common in the Norwich

district of England. It is more common in early and late life than in middle life, and in males than in females. The oxalate calculus as well as the uric acid calculus seems to be favoured by the uric acid or gouty diathesis.

Symptoms.—The condition may be latent all through. Even a large dendritic calculus may be accidentally discovered after death. Or sand or gravel may be passed from the pelvis through the ureter, bladder and urethra, during an indefinite period, without giving rise to symptoms. Or, again, the first symptoms may be those of obstructive suppression, through blocking of the ureter in a case where the opposite kidney is not functionally active.

Renal colic is a very common result of renal calculus. A small calculus gets into the ureter, and not only irritates the delicate mucous membrane, but excites painful spasm of the muscular wall. In a typical case there is pain of sudden onset and intense severity in the lumbar region and flank of the affected side. If often radiates into the scrotum, testis or thigh, and the testicle on the affected side is retracted. The attack may be associated with a rigor, vomiting, profuse perspiration and collapse. The temperature may be elevated. There is frequent, painful micturition, and the urine is red with blood. Diarrhœa may be present. The duration of the paroxysm is from an hour to several days, and the severe pain may come to an end suddenly when the stone reaches the bladder, though a certain amount of aching remains for a time in some instances.

If the stone reaches the bladder, it is usually voided afterwards with the urine. But it may remain in the bladder, and grow there ; or it may become impacted in the urethra, and require removal by operation. It may, however, fail to reach the bladder, and in that case it undergoes arrest in the ureter, or falls back into the pelvis. If the ureter is completely blocked, the kidney atrophies. If the obstruction is incomplete, *hydronephrosis* develops. If the opposite kidney is already functionless, complete obstruction gives rise to *obstructive suppression* as previously described (p. 585). Obviously, if several calculi are present in the kidney, the patient may have repeated attacks of renal colic.

A calculus in the pelvis of the kidney may cause pain in the renal region, either slight or severe, constant or paroxysmal. It is specially seated in the lumbar region of the back, and may radiate downwards and forwards to the lower abdomen and thigh. Tenderness may be present at the back over the hilus of the kidney. Paroxysmal pain of the kind just described may be associated with a rigor, high fever, bloody urine containing much epithelium, and profuse sweating. Osler calls attention to the analogy between this *renal intermittent fever* and hepatic intermittent fever (p. 547), each of which has its origin in calculus; and to the probability that either may occur in severe form without suppuration. It is a striking fact that pain in one renal region may be due to calculus in the opposite kidney, as Newman and others have shown (*reno-renal reflex pain*).

Another symptom of calculus in the pelvis is hæmaturia. The urine is red with blood, and contains not only blood corpuscles, but also transitional epithelium from the pelvis, and sometimes crystals which give an indication of the nature of the stone. The bleeding, like the pain, is apt to be aggravated or induced by exertion.

In cases of old-standing calculus in the pelvis, inflammation supervenes, and this is very often suppurative. It may involve the pelvis itself (*pyelitis*), the substance of the kidney (*pyelonephritis* and *pyonephrosis*), or the tissues around the kidney (*perinephritic abscess*). These complications are described separately.

Diagnosis.—Pain in, and radiating from, the region of the kidney, bloody urine, and subsequent inflammation in the region of the kidney are suggestive of calculus. Pain starting from the region described and radiating into the thigh, shivering, vomiting, retraction of the testicle and hæmaturia point strongly to a calculus traversing the ureter. The matter may be settled by the subsequent evacuation of the stone by the urethra. But the symptoms are often not characteristic. For instance, either pain or bleeding may be the only symptom, or, as already stated, there may be no symptom at all.

The distribution of the pain, the hæmaturia and the re-

traction of the testicle distinguish renal from *hepatic* and *intestinal colic*. The passage of the stone will confirm the diagnosis, but the attack may be due to the passage of blood-clot, a mass of pus, or some other solid substance along the ureter. Moreover, renal colic may be closely simulated by *tubercular disease* of the kidney. In the latter case there may be tuberculosis of other organs, tubercle bacilli may be discovered in the urine, or the family history may be suggestive of tubercle. In calculus, there may be a history of earlier attacks of colic. The X rays may be of service in revealing the presence of a stone in the kidney, and in safeguarding the surgeon from being misled by reno-renal reflex pain. Assistance may also be had from a separate examination of the urine of the two kidneys.

With regard to the nature of the stone, an alkaline reaction of the urine points to a phosphatic calculus ; an acid reaction to uric acid or calcium oxalate. The mulberry (oxalate) calculus is more likely to be associated with severe radiating pain and abundant hæmorrhage than the smooth uric acid stone.

Prognosis.—This is very uncertain, both as regards recurrence of colic and as regards the development of pyelitis and other complications. Suitable treatment, however, may greatly improve the outlook.

Treatment.—In renal colic, chloroform may be given if the pain is intense. In less severe attacks, morphine hypodermically, hot drinks and the hot bath or hot fomentations should be used. A valuable method of treating the attack is that recommended by Wm. Murray. Belladonna is given in large doses at short intervals (say, 20 to 40 drops of the tincture every one or two hours for an adult) till dilatation of the pupil, dryness of the throat and even delirium are produced. This treatment should be kept up not merely till the pain ceases, but till the stone is evacuated by the urethra.

In cases of gravel or renal sand, the urine should be kept constantly alkaline by the administration of citrate or acetate of potassium to the extent of 5 or 6 drachms daily in divided doses. An abundance of plain water or alkaline

mineral water should be drunk every day between meals, and on rising. Moderation in eating, a daily tepid or cold bath, regular exercise, and avoidance of constipation are also to be recommended.

If a renal calculus too large to enter the ureter gives rise to severe symptoms, nephrolithotomy is advisable. When a stone is impacted in the ureter, operative treatment may again be advisable, and if there is suppression of urine, there is practically no hope apart from surgical intervention.

12. PYELITIS, PYELONEPHRITIS AND PYONEPHROSIS.

Inflammation of the renal pelvis may occur by itself (*pyelitis*), or along with inflammation and especially suppuration of the kidney substance (*pyelonephritis*).

Etiology.—(1) *Pyelitis* is often due to extension of inflammation up the ureter, as in cystitis ; in many of these cases the ureter and pelvis are dilated owing to obstruction lower down. (2) Calculi, tuberculosis, parasites, and other local conditions in the pelvis may cause *pyelitis*. (3) Much less commonly, irritating substances brought to the kidneys by the blood and excreted in the urine excite *pyelitis*—*e.g.*, cantharides, turpentine, diabetic sugar, and the poisons of scarlet fever and pneumonia ; some of these may cause *pyelonephritis*.

The causes just mentioned are mostly to be regarded as simply predisposing. The actual exciting agents are usually microbes which reach the pelvis by the lumen of the ureter ; or, as Lindsay Steven has shown, by the lymphatics in the wall of the ureter, the lymph spaces of the capsule and the tubules ; or, thirdly, by the blood-stream. They may reach the substance of the kidney either by way of the pelvis and tubules, or by penetrating from the capsule and the surface of the gland. Among the principal organisms concerned are the *Bacillus coli*, the *Streptococcus*, the *Staphylococcus aureus*, the *Bacillus typhosus*, and the tubercle bacillus.

Morbid Anatomy.—This varies according to circumstances. (1) In *pyelitis* itself the mucous membrane is swollen and greyish, with hæmorrhages and sometimes ulcers. In

calculous pyelitis, the mucosa may be roughened. In specific fevers, a false membrane may be present. The pelvis contains turbid urine with pus or muco-pus and epithelium.

(2) In many cases, and especially where the pyelitis is secondary to cystitis, the infection spreads to the substance of the kidney. Numerous small abscesses form there, and may be seen extending between the pyramids into the cortex. Many may also be found in the cortex close under the capsule. Sometimes such small abscesses coalesce into a large abscess. These conditions are known as *pyelonephritis* or *surgical kidney*.

(3) In many cases, the ureter is obstructed, so that the pelvis and calices become dilated, and the renal tissue atrophies. The kidney is thus converted into a pus-containing sac (*pyonephrosis*). This condition presents some resemblance to hydronephrosis, but the contents of the sac are purulent instead of watery or urinous, and there is more marked inflammatory change in the substance of the kidney. The capsule is generally thickened, and may be adherent to neighbouring parts as well as to the kidney itself.

In accordance with the nature of the primary condition, changes are often found in the ureter and bladder.

Inflammatory affections of the kidney which result from inflammation or obstruction lower down the urinary tract are sometimes termed collectively *consecutive nephritis*.

Symptoms.—In *pyelitis*, the symptoms may be few or may be masked by the cause of the inflammation. There may be dull pain in the back, frequent micturition, and pus and epithelium in the urine. In calculous cases, there may be hæmorrhage. The general condition does not necessarily suffer much. In *pyelonephritis*, however, and in *pyonephrosis*, the constitutional evidences of suppuration appear, including pallor, emaciation, and hectic fever. In pyonephrosis there may be local pain, tenderness and tumour. In such circumstances, the ureter may become blocked for a time, with the result that (in unilateral cases) the pus disappears from the urine, and the tumour enlarges. On the other hand, when the obstruction yields, a large amount of pus is voided, and the tumour disappears.

Diagnosis.—This is often aided by the existence of a possible cause of pyelitis—*e.g.*, calculus or cystitis. The history of the case is very important. The presence of pus in acid urine is very significant if associated with pain or tenderness in the renal region.

In *tubercular pyelitis* (see p. 129), tubercle bacilli may be present in the urine.

In *pyonephrosis*, the nature of the case may be indicated by the temporary absence of pyuria and the simultaneous development of a renal tumour through obstruction of the ureter.

In *pyelonephritis*, general symptoms are more likely to be present than in pyelitis.

In *perinephric abscess*, there may be œdema and fluctuation in the renal region, and the urine may be free from pus.

Prognosis.—Pyelitis in an acute fever is usually of trifling importance, except that it may favour the formation of calculus later on. Pyelitis following the cystitis of gonorrhœa is occasionally fatal, but usually passes off. Calculous pyelitis, even after continuing for many years, may disappear under treatment. In tubercular pyelitis, and in the pyelitis and pyelonephritis which follow obstruction lower down, the outlook is serious; perforation into the peritoneum, waxy degeneration, and exhaustion are the principal risks. Nevertheless, in both calculous and tubercular cases, the disease may undergo arrest; the pus thereafter dries in, becomes infiltrated with lime salts, and remains in the dilated pelvis and calices as a white pultaceous mass.

Treatmnet.—As far as possible, the cause of the condition must be removed. The patient should drink plenty of bland fluids. Tonics and change of air may be of some service. In pyonephrosis, and even where there is no tumour, if the symptoms are distressing and persistent, operation is indicated.

13. PERINEPHRIC ABSCESS.

Etiology.—Suppuration of the cellular tissues around the kidney may be due (1) to local injury, *e.g.*, a blow or strain ; (2) to extension from the kidney substance, pelvis of the kidney, or ureter, *e.g.*, in calculous pyelitis ; or (3) to extension from a more distant lesion, *e.g.*, appendicitis, spinal caries, empyema, etc. The usual cause is calculous pyelonephritis.

Morbid Anatomy.—Occasionally there is a chronic induration of the fatty capsule of the kidney, leading on the one hand to hypertrophy of the adipose and fibrous tissue, and on the other, it may be, to compression of the kidney (*chronic perinephritis*). In most cases, however, suppuration takes place, the pus appearing in several foci. As the pus accumulates, it tends to surround the kidney more or less completely. It has sometimes a fæcal odour through proximity to the colon. The abscess may attain a great size and spread very extensively. It may travel downwards to the groin, it may open into the peritoneum or pleura, or it may burst into the bowel, bladder, or vagina.

Symptoms.—The onset may be insidious or acute. In acute cases febrile symptoms are present, with constipation and concentrated urine. There is pain in the affected region radiating towards the thigh or testis. The hip is often flexed, and the body is bent towards the side of lesion. The affected part is tender.

In a few cases (*simple perinephritis*), resolution takes place without suppuration, and in these no definite tumour develops. But in most cases the inflammation goes on to suppuration (*phlegmonous perinephritis*, *perinephric abscess*), and very often a firm swelling develops in the region of the kidney. This can frequently be recognised as distinct from the spleen and liver. It does not move with the diaphragm. Deep-seated fluctuation may be detected. The overlying tissues may be œdematous, and the pressure on deep veins may give rise to œdema of the lower limb. The urine is likely to contain pus if the suppuration originated

in the kidney or pelvis, and also if the abscess has burst into the urinary system.

Diagnosis.—In *appendicitis* the pain is in front, and is at first over the abdomen generally, and afterwards in the iliac region.

In *lumbago*, the pain is very acute and specially related to the activity of the lumbar muscles. Moreover, the history is different, there is little or no fever, and the symptoms are bilateral.

In *spinal caries*, there is no fever, and the onset is gradual.

In *hydronephrosis*, the temperature is normal, and there is little or no pain.

Prognosis.—After the abscess is opened, either spontaneously or by operation, healing may take place. In other instances, chronic suppuration ensues and leads to death by exhaustion from fever, amyloid disease, etc. Occasionally the abscess causes speedy death by rupturing into the peritoneum or giving rise to septicæmia.

Treatment.—At the onset, rest, aperients, cupping and poulticing are indicated. As soon as the existence of pus is considered to be possible, a free incision should be made for the purpose of exploration and, if need be, further surgical treatment. For chronic suppuration, tonics and change of air are advisable.

14. CYSTIC DISEASE OF THE KIDNEYS.

Cysts in the Kidney.—Apart from the cysts which constitute a common feature of granular kidney, and the rare hydatid cysts, there is often observed a simple cyst which contains a clear watery or colloid fluid. This is generally situated in the cortex and projects from the surface, the rest of the kidney being healthy. It is often solitary. In exceptional cases it forms a large tumour, but as a rule it has no clinical significance. It has been supposed to be due to obstruction of tubules in fœtal life.

Cystic Disease of the Kidneys

(Cystic Transformation or Degeneration of the Kidneys. Congenital Cystic Kidneys).

This disease is of congenital origin, and often causes death within the uterus or shortly after birth, but a considerable number of cases reach adult life. The cystic condition of the kidneys is sometimes associated with a similar condition of the liver, and even of the brain and other organs. Moreover, other congenital abnormalities may be present, such as malformation of the bladder, ureter, etc.

Morbid Anatomy.—The kidneys are greatly enlarged, and consist of a mass of cysts, which vary in size and colour, but are very suggestive in appearance of a bunch of red and purple grapes. The fluid in a cyst is commonly yellowish, but may be reddish or quite dark, owing to the presence of blood. It is, moreover, slightly gelatinous from the presence of colloid matter. The cysts are quite distinct from one another, and are lined by epithelium. The proper tissue of the kidney is atrophied. In some cases, but not in all, cardio-vascular changes are present, probably in consequence of chronic interstitial changes in the surviving renal tissue.

Pathology.—It has been supposed that the cystic disease is due to obstruction of tubules, either by fœtal inflammation, or by the accumulation of colloid material which results from a change in the epithelium.

Symptoms.—Sometimes the kidneys are so large in the fœtus as to interfere with labour. In other cases, the enlargement, if any exist, is not discovered till middle life. In any case the tendency is for both kidneys to undergo progressive increase in bulk with atrophy of secreting tissue until the renal function fails. The tumours may be felt during life. The condition of the urine and cardio-vascular system may resemble that seen in chronic interstitial nephritis. Attacks of hæmaturia may be a symptom.

Death is generally due to uræmia, which is frequently acute, but is sometimes of the latent type seen in obstructive suppression.

Diagnosis.—Symmetrical nodulated tumours in the region of the kidneys, urine with the characters observed in chronic interstitial nephritis, and uræmic phenomena point strongly to this disease.

Prognosis.—As the disease involves progressive failure of function on the part of the kidneys, life cannot be much prolonged after uræmic symptoms appear.

Treatment.—This is the same as that of chronic Bright's disease.

15. TUMOURS OF THE KIDNEY.

The only tumours of clinical importance are sarcoma and cancer. *Sarcoma* is usually secondary, but is sometimes primary, and in the latter case occurs chiefly in intra-uterine life and childhood. The growth is generally round celled or spindle celled, and sometimes contains striated muscle. Primary *cancer* is generally met with after forty years of age, and is more common in males than in females. It is usually encephaloid, and may involve either the whole organ or one part of it, leaving the other part normal. The part involved is enlarged, while retaining the general shape of the healthy organ, and covered by its capsule. It has been supposed that most of the primary cancers and alveolar sarcomas are really growths arising in aberrant portions of adrenal tissue (hypernephromata).

Malignant disease of the kidney has a strong tendency to invade the renal vein and inferior vena cava, and it may thus block the vena cava and both renal veins. Secondary deposits are more common in the lungs than in the liver or lumbar glands.

Symptoms.—The most important are pain, tumour and hæmaturia. A tumour can generally be recognised in the renal region, with the colon in front of it. It is sometimes firm and sometimes almost fluctuant. Pain may be severe, slight, or entirely absent. Attacks of renal colic may be induced by the passage of blood-clots down the ureter. Hæmaturia occurs in about half the cases, and varies much from time to time. Cachexia supervenes in cases of cancer,

and among the less common phenomena of the disease are varicocele, thrombosis of the inferior vena cava with œdema of the lower limbs, invasion of the spine leading to paraplegia, and obstruction of the colon from pressure or invasion.

Diagnosis.—The colon is in front of a renal tumour, whereas it is behind a *large spleen*. Moreover, the latter has a distinctive edge.

The lower edge of the *liver* may be felt, and is likely to be separated from a renal tumour by an area of tympanitic percussion. Both liver and spleen descend with inspiration.

Disease of the colon will be associated with intestinal symptoms, and the results of repeated enemata will give valuable information.

Cystic disease of the kidneys is bilateral.

In *perinephric abscess* there is fever.

In *hydronephrosis* there is not likely to be pain or hæmaturia, and the history of the case may give valuable information.

An *ovarian tumour* begins low down and grows upwards.

Treatment is almost wholly symptomatic. In cases where there is no hæmaturia, exploration would be justifiable, since the tumour might be a sarcoma of the suprarenal body, a growth which is supposed to be more easily extirpated with success than cancer of the kidney.

16. MOVABLE KIDNEY

(Floating Kidney. Nephroptosis).

The kidney, under normal conditions, is fixed in position by the fatty tissue which surrounds it, by the peritoneum in front of it, and by its bloodvessels. In rare cases it possesses a *congenital* mesonephros, so that, like the intestine, it is free to move about in the abdomen. By some writers the expression *floating kidney* is applied to these cases only.

Much more commonly the undue mobility is *acquired* (*movable kidney*). This affects women far more frequently than men, and involves the right kidney much more often than the left. It is attributable to tight-lacing, to relaxation of the abdominal wall by pregnancy, and in the case of the

right kidney to pressure by the liver in inspiration. Emaciation may favour it by diminishing the bulk of the fatty capsule. It is most common in the first half of adult life. It is not confined to women who have borne children. If the kidney can be freely moved about the abdomen, it is often termed *floating kidney*, though there is no mesonephros.

Movable kidney is sometimes a part of the more widespread condition already described under the name of enteroptosis or Glénard's disease.

Symptoms.—Often there are no symptoms. In other cases there is a sense of weight or dragging in the loins or abdomen. Neurasthenic symptoms are common. Occasionally there are attacks characterised by severe abdominal pain, vomiting, scantiness of urine, and perhaps hæmaturia. These paroxysms are not unlike renal colic, and have been called 'Dietl's crises.' They are supposed to be due to strangulation of the kidney by kinking or twisting of the renal vessels. Hydronephrosis, which may be intermittent, occasionally results from kinking of the ureter. Gastric symptoms and dilatation of the stomach have been attributed to dragging on the duodenum.

Diagnosis.—The kidney is often accessible to palpation, and its characteristic form can then be recognised. Firm pressure gives rise to pain, which may be of a sickening character. The lumbar region on the affected side may be less full than the other, and may yield a tympanitic instead of a dull note. The freedom with which the kidney moves in accordance with changes of posture on the part of the patient may be very noticeable.

Treatment.—Many cases require no treatment. If symptoms are present, a flannel binder and pad may be sufficient, the patient applying them before rising in the morning and after pushing the kidney towards its proper position. For symptoms of strangulation, morphine and hot fomentations are indicated. If simpler measures fail, the organ should be stitched to its place in the lumbar region (nephrorrhaphy). It is most desirable to get rid of any neurasthenic condition which may be present.

SECTION VIII

DISEASES OF THE NERVOUS SYSTEM

INTRODUCTION.

Putting out of consideration bloodvessels, lymph channels and supporting structures, the nervous system may be regarded as an aggregate of neurons. A typical *neuron* is a nerve cell with numerous branches. One of these branches is larger than the others, and is continued into the axis-cylinder of a nerve fibre ; whilst the others divide and lose themselves in the surrounding grey matter. The branching processes are called *dendrons* (*dendrites*) ; the axis-cylinder process is called the *axon* (*neurite*). The dendrons conduct impulses towards the cell ; the axon conducts impulses away from it. The axon in its course gives off branches called *collaterals*, and ends by breaking up into ramifications which constitute an *arborisation*. The arborisations of the axon and collaterals are often in close proximity to the dendrons of other nerve cells, but are not structurally continuous with them. An impulse therefore does not pass from one cell to another by an uninterrupted path.

The nerve cell contains a nucleus and fibrillated protoplasm. Among the meshes of the latter there are the angular bodies known as Nissl's granules. These stain deeply with certain basic dyes, such as thionine, toluidine blue, or methylene blue, and a study of their condition throws light upon the state of health of the nerve cell itself.

i. The Motor Path and Motor Symptoms.

The motor path—viz., the path taken by motor impulses in passing from the so-called motor area of the cortex (see

later, Diseases of Brain, Introduction) to the muscles—consists of two segments or series of neurons, upper and lower. The upper segment passes from the cortex to the grey matter of the anterior horn of the cord or the corresponding grey matter within the cranium ; this segment crosses the middle line of the body. The lower segment commences in the grey matter of the anterior horn or in the corresponding grey matter within the cranium, and extends along the corresponding motor nerves to the muscles on the same side.

E.g., (1) in the case of the motor path for the face muscles, the upper neurons have their cells in the cortical grey matter of the lower part of the ascending frontal convolution. The axons extend downwards through the corona radiata, the *genu* of the internal capsule, and the crus cerebri into the pons Varolii. At the middle of the pons they cross the middle line to break up into arborisations in the nucleus of the seventh nerve. The neurons of the lower segment have their cells in that nucleus, while their axons run from these cells in the fibres of the seventh nerve to ramify on the fibres of the muscles of the face.

(2) In the case of the motor path for the muscles of the upper limb, the cells of the upper neurons are in the ascending frontal convolution, at or rather above its middle. The axons pass down through the centrum ovale, the anterior portion of the posterior limb of the internal capsule, the crus and the pons to the pyramids of the medulla, where they mostly cross to the opposite side (see later, Diseases of Spinal Cord, Introduction). They then run down the lateral pyramidal tract to the cervical enlargement, where they enter the anterior horn, and terminate in arborisations which are in close relationship with the motor cells.[1] These cells

[1] This description is allowed to stand because a more accurate one cannot yet be given, and it is in accordance with clinical facts. But it is to be noted that good authorities hold that the fibres of the pyramidal tract (upper motor neurons) do not enter the anterior horn of the cord, but terminate further back, somewhere about the base of the posterior horn, or about the processus reticularis. It has accordingly been supposed that dendraxons (neurons with exceedingly short axons) are interposed between the upper and the peripheral neurons.

are at the upper end of the lower segment, and their axons pass out in the anterior spinal nerve-roots into the nerves of the upper limb and so on to the muscles.

(3) In the case of the motor path for the lower limb muscles, the upper segment begins in the grey matter in the upper part of the ascending frontal convolution. The upper segment ends and the lower segment begins in the anterior horn of the spinal cord at the lumbar enlargement.

In the internal capsule, the fibres of the motor path occupy the anterior two-thirds of the posterior limb ; those for the face being at the bend of the capsule, those for the arm further back, and those for the leg still further back. (The posterior third of the posterior limb is occupied by sensory fibres.) (Fig. 41.)

In the crus cerebri, the motor path occupies the middle two-fifths of the crusta or inferior portion of the crus, the face fibres being innermost, then the arm fibres, and then the leg fibres external to the others. Those axons of the upper segment which are destined for the nucleus of the third nerve cross the middle line in the crus cerebri.

Symptoms of Lesion in the Motor Path.—It is obvious that a destructive lesion of any part of the motor path will interfere with the transmission of voluntary impulses, so that loss of power will result. As a rule the loss of power is associated with other symptoms which vary according as the lesion is in the upper or in the lower segment. Thus interruption of the upper neurons causes weakness without muscular wasting, and without changes in the electrical reactions, but with exaggeration of the tendon-jerks ; whereas interruption of the lower neurons causes, in typical instances, weakness with wasting, with changes in the electrical reactions, and with loss of the tendon-jerks. Cases, however, are common which present features intermediate between these two groups of symptoms.

Irritative lesions of the motor path, but chiefly those which involve the cells of the upper neurons, may give rise to muscular spasm.

Paralysis or loss of power may be due to lesion in any part of the motor path ; it is sometimes due to a lesion of the

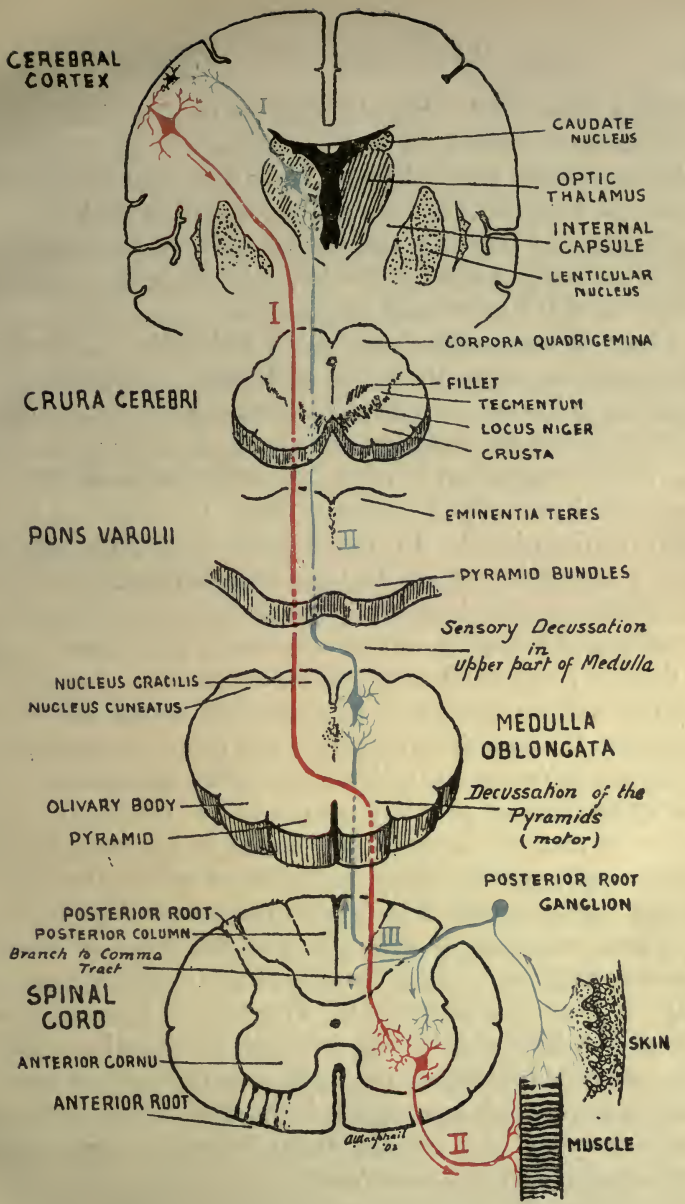

FIG. 33.—DIAGRAM REPRESENTING MOTOR AND SENSORY NEURONS.

The motor path (coloured red) consists of two series of neurons: the upper (I.) extending from the cerebral cortex to the anterior cornu, or to the homologous nuclei of the motor cranial nerves; the lower (II.) extending thence to the muscles. The particular sensory path here figured (in blue) consists of three series of neurons: the lowest (III.) extending from the periphery, by way of the posterior roots and posterior column of the same side, to the nucleus gracilis; the middle (II.) extending from that nucleus across the middle line, and on to the optic thalamus; and the highest (I.) passing thence to the cortex, where its arborisations are seen close to the dendrons of the upper motor neurons. It will be seen that both motor and sensory paths cross the middle line. The lowest part of the illustration shows a reflex arc such as is concerned in the knee-jerk. (See footnote, p. 621.)

muscles themselves. Slighter degrees of loss of power are sometimes described as *paresis*. Paralysis involving one side of the body is called *hemiplegia ;* it is almost always the result of a cerebral lesion, and will be described in detail afterwards. Palsy of one limb is termed *monoplegia*. *Paraplegia* means paralysis of the lower limbs, with or without paralysis of the lower part of the trunk.

Ataxy or *inco-ordination*, though a disorder of muscular movements, is often the result of damage to afferent nerve fibres (as in locomotor ataxy). Almost any movement of the body involves the activity of several muscles, and if the degrees of contraction of these different muscles do not bear the normal proportion to one another, the resulting movement is disordered. In the healthy state, the afferent nerves of muscles convey to the co-ordinating centre in the cerebellum information as to the present condition of the muscles, and founding on this information, the centre is able to direct the muscular contractions necessary to bring about the required movement. If the afferent muscle-nerves are damaged, the co-ordinating centre will receive incomplete or erroneous information, and will obviously be unable to discharge its function properly ; ataxy will be the result. This symptom may also result from disease in the co-ordinating centre itself, or from disease of afferent nerves from other structures than muscles (*e.g.*, from the semicircular canals).

Spasm or excess of muscular contraction is a common motor symptom. Spasm may be *tonic* (continuous) ; or it may be *clonic* (intermittent). *Tremor* is a form of clonic spasm. Sudden and violent involuntary muscular contractions are called *convulsions*. These may be localised, but are often general and accompanied by loss of consciousness. The shortening of a muscle which follows a long existing tonic spasm is known as *contracture*.

ii. The Sensory Path and Sensory Symptoms.

The sensory path is more complex and less understood than the motor path. The lowest neurons have their cells in the ganglia of the posterior roots of the spinal nerves and

of the corresponding roots of cranial nerves. Each cell gives off a process which divides into two. One division passes into a peripheral nerve and so to the periphery (skin, muscle, etc.) ; this may be looked upon as a dendron. The other division passes along the posterior root into the spinal cord and constitutes the axon. After entering the cord, the axon divides into ascending and descending branches. The latter passes down in the posterior column for only a short distance (viz., in the comma tract of the postero-lateral column), and ends in the grey matter of the same side. The ascending branches follow various routes. It is recognised that some of them pass up the posterior columns and end in the nucleus gracilis and nucleus cuneatus of the same side, where the neurons of the second order have their cells. Both ascending and descending branches of the axon first mentioned give off collaterals, and these end in arborisations, some around cells in the anterior horn, thus furnishing the reflex arc for the knee-jerk, etc. ; others round the cells of Clarke's column, whose axons run up in the direct cerebellar tract ; and others, again, round cells whose axons cross to the other side and constitute the antero-lateral ascending tract of Gowers. It is to be noted that the axons of these lowest neurons do not cross the middle line.

The neurons of the second order already mentioned send their axons forwards from the posterior nuclei of the medulla (nucleus gracilis and nucleus cuneatus), across the middle line (decussation of the fillet), to pass in the fillet through the tegmental region of the pons and end in the optic thalamus. From this body there originates a third order of neurons, whose axons end in the cortex, probably in the ascending parietal convolution (post-central gyrus), or in a more extensive portion of the parietal lobe.

These three orders of neurons constitute the simplest and most direct sensory path, but it is known that other paths exist. Thus, a unilateral transverse lesion of the spinal cord causes anæsthesia on the opposite side of the body, from the foot up to a little below the level of the lesion ; a fact which shows that the sensory path in this instance crosses the middle line very soon after entering the cord.

The lowest neurons probably keep to their own side, as in the case of the sensory path first described; while those of the second order send their axons across the middle line almost immediately. The path in this case may be by a long series of neurons, with very short axons which end in the grey matter, or are possibly entirely situated in the grey matter of the cord. This path, like the other that has been described, may be regarded as leading up to the thalamus, whence it also is continued to the parietal cortex. It is conceivable that yet another sensory path may be constituted by Gowers's antero-lateral ascending tract, whose axons, after crossing the middle line and running upwards in the cord, pass to the cerebellum by way of its superior peduncle.

The different kinds of sensory impressions (touch, pain, temperature, etc.) do not all travel by the same routes, but this point will be further alluded to in connection with syringomyelia.

Sensory Symptoms.—The various kinds of sensation may be lost or disturbed apart from one another, so that they must be tested separately. Those which specially require attention are tactile, painful and thermal sensations, the sense of posture, common muscular sensibility (recognised by compressing the muscles by the fingers), and the stereognostic sense (recognition of the shapes of objects by handling them). *Anæsthesia* means loss, and *hyperæsthesia* exaggeration of sensation, and of the sense of touch in particular, but thermal anæsthesia and hyperæsthesia are also expressions in use. *Paræsthesia* means disordered sensation—*e.g.*, tingling or a feeling of 'pins and needles.' *Analgesia* and *hyperalgesia* mean respectively absence and exaggeration of the sense of pain. *Allochiria* is a sensory abnormality characterised by the patient referring a sensation to a different part from that to which the stimulus was applied—*e.g.*, to the opposite side of the body.

Delay in the conduction of sensory impressions is sometimes observed, so that a distinct interval, amounting perhaps to many seconds, may elapse before the patient is aware that a stimulus has been applied to his skin.

iii. Reflex Action.

Changes in reflex action are common and important results of disease in the nervous system. Loss may be due to a lesion in any part of the reflex arc, or in the muscle to which the efferent neurons are distributed. Three groups of reflexes may be mentioned : (1) Of the *tendon-reactions*, *deep reflexes* or *muscle reflexes*, the best known is the *knee-jerk*, *knee phenomenon*, or *patellar tendon reflex*, which is always present in a state of health. When the knee is slightly flexed—*e.g.*, by being thrown across the other knee when the patient is sitting—a blow upon the patellar tendon causes a contraction of the quadriceps extensor, so that the foot is jerked forwards. The tension induced in the muscle by flexing the knee causes by a reflex process an increase in its mechanical irritability. The result is that when the tension is suddenly increased still further by the blow on the tendon, the muscle contracts. The centre for this reflex is in the third and fourth lumbar segments of the cord.

The *ankle clonus*, *foot clonus* or *foot phenomenon* is not normally present. It consists of clonic contractions of the muscles connected with the tendo Achillis induced and kept up by passive flexion of the ankle. The centre is in the first and second sacral segments. Occasionally other muscles—*e.g.*, the rectus femoris—become the seat of clonus. *Myotatic irritability* is a convenient expression introduced by Gowers to indicate that form of muscular irritability which manifests itself in the knee-jerk and other so-called deep reflexes. This irritability is the result of a reflex action whose centre is in the spinal cord, and which is probably also the cause of ordinary muscular tone.

Exaggeration of the knee-jerk and presence of ankle clonus result from disease of the upper motor neurons which, as is commonly supposed, withdraws control from the lower neurons. Loss of the knee-jerk may be due to disease of the afferent or efferent muscle nerves, as in multiple neuritis ; of the centre in the cord, as in myelitis ; or of the muscles themselves, as in muscular dystrophy. If the knee-jerk cannot be obtained at first, an effort should be made to

reinforce it by making the patient exercise muscles in some other part of his body while the test is being made. Thus, he may clasp his hands and pull his fingers strongly apart. At the same time he should turn his eyes away, and be engaged if possible in conversation.

(2) The *superficial* or *cutaneous reflexes* are induced by stroking, pinching or otherwise stimulating the skin. Thus, when the sole is stroked the muscles of the toes contract, constituting the *plantar reflex*. The most important point to note in connection with this reflex is the behaviour of the great toe. In health it is flexed as a rule, but in hysterical and other functional disorders it often remains motionless. On the other hand, in degeneration of the pyramidal tract (upper neurons) it is extended. This extensor type of plantar reflex is known as *Babinski's sign*, and when met with in clinical work points, as a rule, to organic disease involving the upper neurons. It is sometimes obtainable, however, for some time after an attack of convulsions, and is said to occur in strychnine poisoning and in tetanus. Moreover, it is a normal phenomenon in infants who have not begun to walk. It must be borne in mind that absence of Babinski's sign does not exclude disease of the upper neurons. The centre for the plantar reflex extends from the fifth lumbar to the second sacral segment of the spinal cord.

The *cremasteric reflex* is characterised by retraction of the testicle on stroking the skin of the inner aspect of the thigh, or on pressing over the adductor foramen. The centre is in the three highest lumbar segments. The *gluteal*, *abdominal*, and *epigastric reflexes* are characterised by contraction of the muscles in the regions named, when the overlying skin is stroked. The *conjunctival reflex*, and the *dilatation of the pupil* which follows stimulation of the skin of the neck, may also be included in this class.

(3) The *organic reflexes* are connected with the functions of micturition, defæcation, deglutition, contraction of the pupil to light, etc. The centres for the bladder and rectum are in the sacral region.

iv. Nutrition.

Disturbances of nutrition are common in disease of the nervous system. The nutrition of a muscle depends on its connection with the cells in the anterior horn of the cord from which its motor nerve fibres are derived, and if the lower neuron is destroyed or divided the muscle becomes flabby and undergoes atrophy. With the exception just mentioned—viz., muscles—the various tissues (skin, bones, joints, etc.) have their nutrition controlled through nerve fibres belonging to the posterior spinal nerve roots. Thus, muscular wasting is met with in disease of the spinal cord and motor nerves, *e.g.*, myelitis, anterior poliomyelitis and neuritis. Nutritive changes in the skin, joints, etc., are met with in diseases of the cord and afferent nerves, *e.g.*, myelitis, locomotor ataxy and neuritis.

v. Electrical Reactions.

In a condition of health, *nerves* are stimulated by both the continuous and the interrupted current. In the case of a sensory nerve, sensation is induced ; in the case of a motor nerve, muscular contraction is induced.

In the case of *muscles* contraction continues as long as the faradic current is applied, whereas galvanism causes contraction only on making, breaking, or altering the strength of the current. Moreover, in connection with the galvanic current it is to be noted that contraction takes place most readily when the negative pole or kathode is placed over the muscle, and the circuit is completed or closed (kathodic closing contraction, KCC). With a stronger current, contraction may be obtained at the positive pole or anode, both on making or closing (ACC) and on breaking or opening the circuit (AOC). Lastly, with a still stronger current, contraction is obtained with the kathode on opening (KOC). Accordingly, the normal polar arrangement is :

$$1.\ \text{KCC}\ ;\ 2.\ \left\{\begin{matrix}\text{ACC}\\ \text{AOC}\end{matrix}\right.\ ;\ 3.\ \text{KOC}.$$

In disease the order may be : 1. $\begin{cases} \text{KCC} \\ \text{ACC} \end{cases}$; 2. AOC ; 3. KOC ;
or 1. ACC ; 2. KCC ; 3. KOC ; 4. AOC.

The points at which muscles are most effectively stimulated by electricity are spoken of as their *motor points*.

When the motor nerve fibres distributed to a muscle are degenerated they are unable to convey motor impulses to the muscle. Degeneration results from the nerve fibres or axons being cut off from the cells of which they are processes, and which control their nutrition. This severance may be brought about by section or other forms of trauma, or by inflammation.

When motor nerves are degenerated, not only is there loss of voluntary power with wasting in the muscles to which the damaged nerves are distributed, but a series of changes in the electrical phenomena of the nerves and muscles may be observable, namely :

(1) Loss of response on the part of the nerves to either faradism or galvanism.

(2) Loss of response of the muscles to faradism.

(3) Increased response of the muscles to galvanism.

(4) Qualitative or polar changes as enumerated above—*e.g.*, ACC as soon as, or sooner than KCC.

(5) Sluggish instead of quick muscular contraction to galvanism (attributable to the muscle fibres being stimulated directly and not through the medium of their motor nerve endings as in health).

When all these changes are present, they constitute the *reaction of degeneration* (R.D.). In actual cases, however, one or several changes may be present without the others.

DISEASES OF NERVES.

i. Neuritis in General.

Neuritis may involve one or many nerves, and may be acute or chronic. The inflammation may attack chiefly the sheath of the nerve (*perineuritis*) or the interstitial tissue (*interstitial neuritis*) ; or the nerve fibres themselves may be primarily involved (*parenchymatous neuritis*).

Etiology.—The *isolated* form is often due to a local cause, such as a wound or prolonged pressure ; or it may be due to extension of inflammation from some neighbouring focus of disease, or to exposure (*rheumatic neuritis*), or to a morbid blood state, such as gout. In the case of morbid blood states, however, it is probable that some local condition is a contributory cause. Isolated neuritis is usually interstitial. *Multiple neuritis*, on the other hand, is generally due to a morbid blood state, and is parenchymatous in its nature.

Morbid Anatomy.—In *acute interstitial neuritis* the affected nerve is red, swollen and infiltrated with round cells. The nerve fibres may be scarcely altered. In *parenchymatous neuritis* the changes in the fibres are primary, and closely resemble those observed in the degeneration which follows section of a nerve. The myelin breaks up into segments and afterwards into small globules, which are ultimately removed. The axis cylinders become broken where the myelin sheath is broken, and they too ultimately disappear. The nuclei of the sheath increase in number. In *chronic interstitial neuritis* there may be wasting of the white substance, and considerable increase of the fibrous-tissue elements without much damage to the axis cylinders.

When the conductivity of a nerve fibre is completely interrupted by inflammation in the way which has been indicated, the fibre degenerates down to its peripheral ending (*secondary degeneration*). Modern methods of examination suggest that changes take place as far up the neuron as its trophic cell in the anterior horn, though this change is trifling in comparison with that which takes place distally to the lesion.

Occasionally a neuritis spreads up a nerve (*neuritis migrans*, *ascending neuritis*), and on reaching a plexus excites inflammation in other nerve trunks.

ii. Isolated Neuritis.

Symptoms.—In *acute* isolated neuritis there is little or no constitutional disturbance. The chief symptom is pain in the nerve and often in its area of distribution. The pain may be severe, radiating and worse at night. The nerve, if

accessible to the finger, may be felt to be swollen. It is tender owing to involvement of its *nervi nervorum*, which are branches given off by itself. Placing the limb or other part in such a posture as to cause tension on the nerve increases the pain. The overlying skin is hyperæsthetic, and may even be red and œdematous through involvement of the vaso-motor nerve fibres. Patches of anæsthesia may be detected. The muscles supplied by the nerve lose their power, undergo wasting, become flabby and show fibrillary twitchings. They are tender on pressure, and their electrical reactions are altered.

In *chronic* cases the changes are similar in kind, but less in degree, and in addition trophic lesions of the skin and joints are frequent. The skin becomes red, smooth, thin and shiny ('glossy skin' of Paget). The subcutaneous tissue also wastes, so that in the case of the fingers the ends of the digits are pointed. The hair and nails may fail to grow properly. Adhesions may develop in the joints, interfering with movement.

Diagnosis.—This depends on the localisation of pain in the territory of a nerve trunk; tenderness of that nerve; constant presence of the pain, though this varies in severity; and signs of diminished functional capacity of the nerve—*e.g.*, anæsthesia, paralysis, muscular wasting and other trophic changes.

Prognosis.—In acute cases the severe symptoms commonly persist for some weeks, and the case then tends to assume a chronic aspect, the pain perhaps continuing for months. The tendency is for the disease finally to pass off. Cases which depend on morbid blood states, such as gout, may be very obstinate. Occasionally inflammation ascends in a nerve and spreads to other nerves, and even to the spinal membranes and cord.

Treatment.—The cause should be removed if possible. At the outset the patient should get a mercurial purge and a diaphoretic. Rest is necessary for the inflamed structures. Fomentations should be applied locally, and cocaine hydrochloride should be injected over the nerve to relieve the pain ($\frac{1}{5}$ increased to $\frac{1}{2}$ grain). If the muscles waste much, they

should be treated with galvanism from time to time till the nerves recover.

In chronic cases counter-irritation and galvanism over the affected nerve, with general tonic treatment, are indicated. For obstinate neuralgic pain, galvanism may be employed with the positive or sedative pole over the painful part. On the other hand, faradism might be applied to the overlying skin by way of counter-irritation.

iii. Multiple Neuritis

(Polyneuritis. Peripheral Neuritis).

Multiple neuritis is primarily an inflammation of the nerve fibres (parenchymatous). It is symmetrical, since the poison is conveyed to the nerves by the blood. The inflammation is most intense in the peripheral portions of the nerves, since these are furthest removed from their trophic centres.

Etiology.—The agents which may damage the nerves are of many kinds, and include metallic elements such as lead, arsenic and silver; organic compounds like alcohol, carbon bisulphide, carbon monoxide and naphtha; the toxins of infections like beri-beri, diphtheria, small-pox, measles, influenza and septicæmia; the morbid blood states associated with diabetes or induced by exposure; and the reduced vitality associated with cachectic and senile conditions.

Some of these poisons show a remarkable power of selecting particular groups or classes of nerves. Thus, alcohol attacks the mixed nerves over the body generally, but especially in the lower limbs. Lead in many cases confines its operations to a certain group of motor nerve fibres in the upper limbs. The toxin of diphtheria attacks by preference the mixed nerves of the palate, and after these the nerves of the ciliary muscles, and after these again the nerves of the legs.

Symptoms.—*Alcoholic neuritis* is one of the most common varieties of the disease, and may be described here as a type representing some of the others. Cases are met with in the two sexes with nearly equal frequency, but as alcoholic excess is not so common among women as among men, it must be inferred that women are more susceptible than men

to this particular result of alcoholism. One explanation probably is that women commonly drink less at a time but more steadily than men, so that they escape the interruptions which follow severe bouts of intoxication, and thus keep their nerves constantly soaked with diluted alcohol. The onset is usually gradual, and is indicated by pains in the legs with tingling or numbness. When the affection is fully developed, the pains are severe ; ankle-drop is present owing to weakness of the muscles in front of the leg ; and walking or standing is impossible, owing to the loss of power, the inco-ordination and the tenderness of the plantar nerves. Before the power of walking is actually lost, a high-stepping gait may be observed, the patient in this way endeavouring to prevent the feet from scraping the floor. In a few cases the alcohol poisons chiefly the afferent muscle nerves, so that the principal symptom is inco-ordination. This variety is known as *alcoholic pseudo-tabes* or *ataxic polyneuritis*. (Pseudo-tabes has occasionally been met with also in arsenical poisoning.) The skin is livid and abnormally sensitive. Tactile sensation is impaired. The nerves are tender, and the muscles are still more so. The knee-jerks are lost. The limbs may be œdematous. The muscles waste, and their electrical reactions are altered.

The upper limbs tend to suffer in a similar manner, but later and in less degree. Mental symptoms are common, and include general enfeeblement, loss of memory, hallucinations and delirium. One of the most striking features of the mental derangement is the tendency shown by the patient to describe in a circumstantial manner experiences which are quite imaginary. The combination of these mental symptoms with those of multiple neuritis is sometimes known as *Korsakoff's disease*.[1] In severe cases the phrenic nerves may

[1] *Korsakoff's disease* or *syndrome* is rather more common in men than in women. Though almost always due to alcohol, it appears to be a result, in a few cases, of enteric fever, digestive disturbance and other ailments. The particular kind of *paramnesia* or perversion of memory which is one of the type-symptoms of the syndrome, viz., the false reminiscences and the forgetfulness of recent events, is not pathognomonic, since it may result from coarse intracranial lesions such as cerebral softening.

be involved with resulting paralysis of the diaphragm, and the vagi with resulting tachycardia. Even an oculomotor nerve may be involved, but as a rule the cranial nerves escape. The bowels and bladder are unaffected, though incontinence may result from the mental condition.

If care is not taken, deformity may result from the limbs becoming fixed in some unsuitable position.

Acute febrile polyneuritis has been described as following exposure to cold or exertion, or as coming on spontaneously like an acute fever. Certainly it is sometimes impossible to discover any satisfactory explanation of an attack of severe multiple neuritis. In this variety the onset is characterised by shivering, pains in the head, back and limbs, and considerable elevation of temperature. Then there develop signs of damage to motor and sensory nerves in the limbs, and it may be in the trunk also. It is doubtful if this disease should be regarded as different from the polyneuritis which is sometimes induced by the poison of influenza. (See also chapters on Diphtheria, Beri-beri, Lead Poisoning, and Arsenical Poisoning.)

Diagnosis.—Among the most important points in the diagnosis of alcoholic and acute febrile multiple neuritis are the symmetry of the symptoms, the involvement of both motor and sensory nerves, and the tenderness of the nerves and muscles. In cases where the affection is chiefly one of afferent nerves, the escape of the sphincters and the preservation of the pupillary light-reflex are of value as pointing against spinal cord disease; while muscular tenderness is also in favour of neuritis.

Prognosis.—After weeks or months, if the cause is removed, improvement generally sets in and recovery slowly takes place. But acute disease—*e.g.*, pneumonia or bronchitis—may carry off the patient, and this risk is naturally increased if the phrenic, intercostal, and other respiratory nerves are inflamed. Phthisis is another common cause of death. Or death may result from other lesions due to alcoholism, *e.g.*, cirrhosis of the liver. In some cases where life is not threatened, there may be permanent disablement, either because the alcohol has not been entirely discontinued,

or because the nerves have been irretrievably damaged, or because of some deformity which has developed.

Treatment.—Alcohol must be entirely withdrawn, and in private cases this means that the patient must be put under the charge of a reliable nurse. Rest is of course essential, and an abundance of good food is important. For a time the treatment is chiefly symptomatic. Thus, severe pain must be relieved by fomentations, anodyne liniments, injections of cocaine, etc. From the very outset, it is essential to guard against deformities such as are very apt to result from foot-drop and constant flexion of the knees. Sandbags and other apparatus may be necessary for this purpose. Galvanism should be applied to the paralysed muscles to maintain their nutrition till the nerves recover. In the later stages, after the pain has passed away, massage may be employed to improve the condition of the muscles and to get rid of deformities, but regulated voluntary exercises are the best agency for this purpose. Strychnine, iron, cod-liver oil and other tonics may also be employed.

In other than alcoholic cases, other measures may be indicated, in addition to the removal of the cause. Thus Gowers recommends large doses of tincture of the perchloride of iron for septicæmic cases (20-30 minims, three or four times a day).

iv. Tumours of Nerves.

Tumours of nerves are called *neuromata*. In a *true* neuroma there is an overgrowth of nerve fibres, either medullated or non-medullated. In the *false* neuroma the new-formed tissue is not nervous, but fibrous, sarcomatous, or of some other kind. In syphilis and leprosy, growths of a specific kind may form on nerves. The *plexiform* neuroma is a rare growth, which consists of interlacing cords, and is usually of congenital origin. The *painful subcutaneous tumour* (*tubercula dolorosa*) is commonly looked upon as a growth on the ending of a sensory nerve, but appears in some cases at least to be a myoma. *Amputation* neuromata (*bulbous nerves*) are enlargements which develop on the ends of the divided nerves after amputations. Growths on nerves

are sometimes present in enormous numbers. *Recklinghausen's disease* is characterised by multiple neurofibromata. There are tumours in or under the skin, and on the nerves. Brown pigmentation of the skin, nævi, and changes in the intellectual functions and in speech may also be present, in addition to motor and sensory symptoms induced by the tumours on the nerves. The disease is sometimes congenital.

Neuromata, especially multiple true neuromata, may give rise to no symptoms. Often, however, they are tender, and are the seat of pain which may radiate. Occasionally there is paræsthesia, anæsthesia, loss of power, or reflex spasm.

The **treatment** is purely surgical, except in the case of specific new formations; but unless considerable suffering is caused by the growth, it should, if of a simple nature, be left alone.

v. Diseases of the Cranial Nerves.

(1) Olfactory Nerve.

The sense of *smell* results from stimulation of the olfactory nerves through the anterior nares. *Flavour* is due to stimulation through the posterior nares. In testing the sense of smell, it is necessary to avoid the use of substances such as ammonia, which stimulate the fifth nerve. Phials containing cotton-wool moistened with the oils of peppermint, anise, cinnamon, cloves, etc., are suitable as tests.

The olfactory nerves pass from the olfactory mucous membrane to the olfactory bulbs. The olfactory tract on each side passes backwards from the bulb, and is continued into two roots, mesial and lateral. The mesial root is continued into the external longitudinal stria, and thus runs backwards on the surface of the corpus callosum, and then forwards through the hippocampal gyrus to the uncus of the same side. The lateral root runs to the uncus of its own side, and is then probably continued by way of the anterior commissure to the internal capsule (sensory crossway) and cortex of the opposite side.

Loss of smell (*anosmia*) is most commonly due to disease of the nasal mucous membrane. Paralysis of the fifth nerve

may cause it by inducing dryness of the mucosa. Fractures of the cribriform plate, meningitis, intracranial tumours, and congenital absence of the olfactory nerves are other causes of anosmia. Unilateral loss may occur in disease of different parts of the brain and in hysteria.

An exaggerated sense of smell (*hyperosmia*) occurs in hysteria and insanity.

Perversion of the sense (*parosmia*) occurs in rare instances as a result of irritation of the nerve or centre.

Subjective sensations of smell may occur in tumour or other disease of the nerve or centre. They may also occur as an aura in epilepsy, and as an olfactory hallucination in insanity.

In disease of the olfactory system the **prognosis** is seldom good, and the principal **treatment** is removal of the cause if this is possible.

(2) Optic Nerve.

The optic nerves pass back to the chiasm, where a partial decussation takes place. The fibres from the left half of each retina pass into the left optic tract, whilst those from the right half of each retina pass into the right optic tract. Each tract passes back to three ganglia on the same side—the pulvinar or posterior part of the optic thalamus, the external geniculate body, and the anterior quadrigeminal body. From each of these bodies fibres pass, by the posterior part of the internal capsule and the optic radiations, to the mesial surface of the occipital lobe of the same side, close to the calcarine fissure. The macular or central region of each retina sends fibres into both optic tracts. The right halves of the retinæ correspond to the left halves of the visual fields, and *vice versâ*.

A lesion of one optic nerve causes corresponding impairment of vision in the eye with which it is connected. A lesion invading or compressing the middle portion of the optic chiasm—viz., the crossing fibres from both eyes—causes blindness of the nasal halves of both retinæ or the temporal halves of both visual fields (*temporal hemianopia*).

This may be, *e.g.*, a tumour of the pituitary body, as in acromegaly. Very rarely the two lateral margins of the chiasm are damaged while the middle portion escapes. The uncrossed fibres therefore suffer, and blindness is produced in the temporal halves of the retinæ or nasal halves of the fields (*nasal hemianopia*). A lesion of the optic tract, thalamus or occipital lobe, causes *lateral* or *homonymous hemianopia*. In this condition the left halves of the two retinæ (right halves of the two fields) suffer when the lesion is on the left

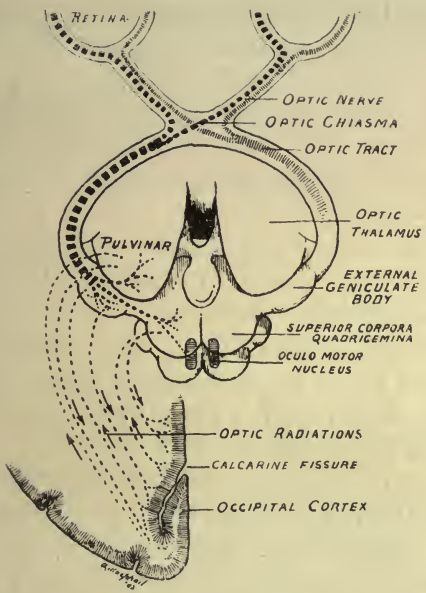

FIG. 34.—SHOWING THE COURSE OF THE OPTIC FIBRES.

side, and the right halves of the retinæ when the lesion is on the right side. The macular region escapes, however, so long as only one optic tract suffers, since it is connected with both sides of the brain.

Sometimes the hemianopia is incomplete, only part of a half field being lost, but the corresponding parts suffer on both sides.

Lateral hemianopia may be produced by disease of the optic radiations or of the cuneus. It then resembles that

due to disease of the tract, except with regard to Wernicke's *hemiopic pupil reaction*. If the lesion is confined to the optic radiations or cortex, the mechanism for the pupillary light-reflex (retina, optic nerve and tract, primary optic ganglia and third nerve) will be intact. By taking great care it may be possible in such a case to throw a light on the blind half of the retina, with the result that contraction of the pupil occurs. If, however, the lesion is in the primary optic ganglia or tract, the light reflex will be lost so far as the blind half of the retina is concerned.

Occasionally there is hemianopia for colour when there is none for white light (*hemiachromatopia*).

Lesions of both occipital lobes cause, in rare instances, *double lateral hemianopia*, which may amount to complete blindness.

There are no ophthalmoscopic changes related to hemianopia.

Hemianopia may be functional (frequently in migraine, rarely in hysteria), or due to organic brain disease (*e.g.*, softening, hæmorrhage, or tumour). It is often associated with hemiplegia. When due to actual destruction of brain tissue, it is not likely to be recovered from ; but if there is simply compression of nerve fibres by a recent hæmorrhage, the symptoms will pass off.

Optic Neuritis (*papillitis*), or inflammation of the intra-ocular termination of the optic nerve, is a common symptom in cerebral tumour, and also occurs in meningitis and cerebral abscess ; sometimes in morbid blood states, as in Bright's disease, lead poisoning and anæmia ; and sometimes after specific fevers. The optic disc in a severe case is concealed by a large red striated swelling, over the margins of which the bloodvessels are seen to bend. Hæmorrhages are often present. The condition is usually bilateral. The inflammation may be severe before the patient notices any loss of vision.

Optic Atrophy may be primary, secondary or consecutive. (1) The *primary* form occurs as an isolated lesion or in association with locomotor ataxy, general paralysis of the insane, or insular sclerosis. Pallor, shrinking and stippling

of the disc are the principal ophthalmoscopic signs. The pallor has often a bluish-grey tint, and for a long time the vessels are unaltered. As the atrophy advances there is concentric diminution of the visual fields. The patient complains of progressive failure of vision.

(2) *Secondary* atrophy is due to a lesion in the optic nerve behind the eye, or in the chiasma. The ophthalmoscopic signs of atrophy follow the impairment of vision instead of keeping pace with it.

(3) *Consecutive* (*neuritic* or *post-neuritic*) atrophy is that variety which follows optic neuritis. The shrinking of the cicatricial tissue may cause greater damage to the nerve fibres, and consequently greater impairment of vision, than the preceding acute inflammation. In this form the disc may be white like chalk. It is filled in, and has often an irregular margin, whilst the vessels may be narrowed owing to compression by the new-formed tissue.

Hereditary Optic Atrophy (*Leber's atrophy*) is a rare disease which may occur in several members of a family and in several generations. Like certain other developmental diseases, it is transmitted mostly to males and through females. The ophthalmoscope shows slight neuritis in the early stage and atrophy later on, but the condition seldom leads to complete loss of vision.

Amblyopia (blunting or dimness of vision) and Amaurosis (darkness of vision, viz., complete blindness) are old terms applied in pre-ophthalmoscopic days to defects of vision, where no signs of disease could be recognised by the physician. They are now used rather loosely to indicate visual defects which are not associated with marked ophthalmoscopic changes. Impairment of vision with signs of optic atrophy is sometimes admitted to this category. Amblyopia and amaurosis may be due to changes in the optic nerve or in the brain, or to a morbid blood state (*e.g.*, uræmia); or they may be functional in their origin.

(3, 4, and 6) Motor Ocular Nerves.

The external muscles of the eyeball are innervated by the third, fourth, and sixth nerves. Of the internal muscles the

iris is supplied by the third and the sympathetic, and the ciliary muscle by the third.

The nucleus of the third nerve consists of groups of cells in the grey matter under the floor of the anterior part of the Sylvian aqueduct. The nucleus of the fourth nerve is nearly continuous with the posterior end of the third nucleus. The nucleus of the sixth is some distance further back, being situated in the pons underneath the floor of the fourth ventricle. The sixth nerve supplies the external rectus muscle ; the fourth supplies the superior oblique, and the third supplies the remaining external muscles of the globe as well as the levator palpebræ superioris. The third nerve supplies the sphincter of the iris and the ciliary muscle. The sympathetic innervates the dilating mechanism of the pupil.

For the purpose of conjugate movement of the two eyes, each sixth nucleus innervates the external rectus of its own side, and sends fibres by way of the posterior longitudinal bundle and opposite third nucleus to innervate the internal rectus of the opposite eye (Fig. 35).

Palsy of Ocular Nerves.—This may be due to compression by tumours, or to syphilitic, tubercular, or other disease of the membranes, cranium or brain. New formations in the posterior part of the brain may squeeze the sixth nerve between the pons and the cranial base. In the orbit the nerves may suffer from tumour or from rheumatism, viz., the blood state which follows exposure. The diphtheritic toxin is another cause of ocular palsy. In certain families there is a tendency to show defective power of the external ocular muscles, and particularly of the obliqui, the superior and inferior recti, and the elevators of the lids. These palsies depend upon absence or poor development of the muscles, and this defect in turn has been supposed to depend upon defects in the cells of the motor ocular nuclei.

The symptoms of ocular palsy include loss of movement, strabismus or squint, diplopia or double vision, secondary deviation of the sound eye, and erroneous projection of the visual field.

(1) *Loss of Movement.*—If the palsy is complete, the eye

cannot be moved by the affected muscle, but stops at the mid-position. After a time the eye becomes drawn in the opposite direction by contraction of the opposing healthy muscle.

(2) *Strabismus* (*squint*) is non-correspondence of the visual axes, and is easily observed if the palsy is severe. It may be convergent or divergent. This *paralytic* form of strabismus must be distinguished from *concomitant* strabismus, the squint which so commonly sets in in early life. The dis-

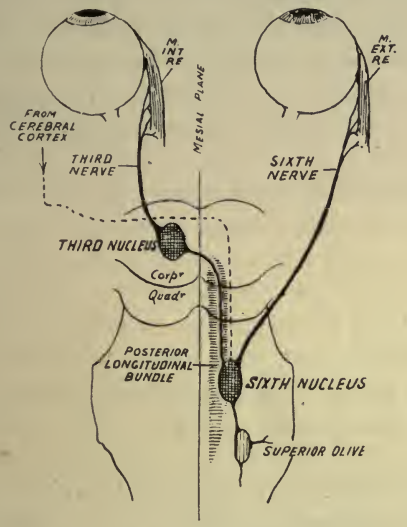

FIG. 35.—MECHANISM FOR THE CONJUGATE MOVEMENT OF BOTH EYEBALLS TOWARDS THE RIGHT.

tinction between the two is made by observing the secondary deviation.

(3) *Diplopia* (*double vision*) results from the images formed on the two retinæ not being superimposed in the normal manner; the two eyes see separate images instead of one composite image. In cases of slight weakness the muscle involved is detected by means of the separate images (see below). The blurring of vision caused by the diplopia may give rise to *giddiness* on moving about.

(4) *Secondary Deviation.*—If the patient gazes with the sound eye at an object in front, the affected eye usually

deviates from the position of parallelism with the other (*primary deviation*). If now the patient fixes the object with the affected eye, it will be found, in recent cases, that the healthy eye deviates from parallelism to a still greater extent (*secondary deviation*). In concomitant squint, previously mentioned, the secondary is equal to the primary deviation.

(5) *Erroneous Projection.*—If the patient, in order to look at a certain object, has to put the weakened muscle into activity, he will project the image of the object too far in the direction of activity of that muscle. *E.g.*, if the right external rectus is palsied, and the patient tries to touch an object in front and to the right, his finger will pass to the right of the proper place. In the effort to rotate the eye by the use of the weak muscle, an abnormal amount of energy is put forth, and this causes the patient to overestimate the distance at which the object is from the mid-position.

Paralysis of the Third Nerve is more frequently partial (*i.e.*, confined to one or several of the muscular branches) than complete. When it is complete, there is *ptosis* (see p. 646) with synergic overaction of the frontalis muscle, and all the external ocular muscles are paralysed except the external rectus and superior oblique. Moreover, the sphincter of the iris and the ciliary muscle are also paralysed.

Paralysis of the Fourth Nerve.—The resulting palsy of the superior oblique muscle may be difficult to detect except by studying the diplopia. An object is seen double when the patient looks downwards.

Paralysis of the Sixth Nerve causes loss of function of the external rectus muscle, so that the eye cannot be rotated outwards.

Diagnosis of the Muscle Involved.—The first thing is to ascertain the relations between the eyes and the images.[1] If the image to the patient's right corresponds

[1] This may be done by putting a piece of coloured glass in front of one eye, so that one of the images is coloured or dimmer than the other ; or the hand may be placed in front of one eye first, and then in front of the other. The observations should be repeated until the results are quite unequivocal.

to his right eye, the diplopia is *homonymous*, and is due to convergence of the visual axes. If his right eye corresponds to the image on his left, the diplopia is *crossed*, and is due to divergence of the axes.

For the rest of the investigation two rules proposed by Landolt are useful guides. It is to be noted that the so-called *false* image is that which corresponds to the affected eye, the *true* image corresponding to the healthy eye. In accordance with the first rule, that *eye* is affected in the direction of *the image of which* the diplopia increases. *E.g.*, if the diplopia becomes greater when the patient looks to his left, the image on the left side corresponds to the affected eye, viz., the left eye if the diplopia is (as previously ascertained) homonymous, the right eye if the diplopia is crossed.

According to the second rule, that *muscle* is affected which would give to the eye the position and direction of the false image. *E.g.*, let it be assumed that by the first rule, the right eye has been found to be at fault. If then the false image is found to be (*a*) above the true, (*b*) to the left of the true, and (*c*) with its upper end tilted to the left, the superior rectus muscle is at fault, since it turns the right eye upwards, rotates it to the left, and tilts the upper end of its vertical meridian towards the left.

To those who can bear in mind the physiological action of the ocular muscles, these rules of Landolt's are simple and easy of application. To others the ingenious and yet simple diagrams of Dr. Louis Werner may be recommended. The one (Fig. 36) applies to the superior and inferior recti ; the other (Fig. 37) to the obliques. The action of the external and internal recti is so simple that no such device is necessary. The continuous lines represent true images, and the interrupted lines false images. *E.g.*, it will be seen that when the right inferior rectus is paralysed, the false image is lower than the true and has its lower end tilted away from the true image. The diplopia is crossed, since the false image is to the left of the true, and the diplopia is perceived in the lower half of the motor field—*i.e.*, when the patient looks downwards. It is to be noted that in the diagram for

the obliques, the inferior muscles are put at the top, and the superior muscles at the bottom.

Ptosis (drooping of the upper eyelid) is one of the most common manifestations of partial palsy of the third nerve. It is often congenital (see p. 642), and may then be unilateral or bilateral. It occurs in hysteria. Transient ptosis may be present for some time after waking in the morning, especially in delicate women. Ptosis is sometimes observed in locomotor ataxy. Loss of tone of the sympathetic may cause slight drooping of the upper lid; and in rare cases ptosis results reflexly, from irritation of the fifth nerve.

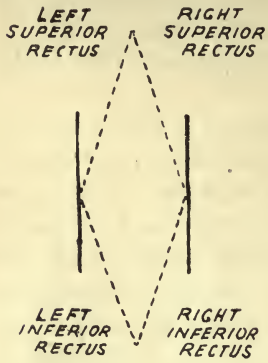

Fig. 36.—Diagram for the Investigation of Diplopia due to Paralysis of a Superior or Inferior Rectus Muscle of Either Eye. (After Werner.)

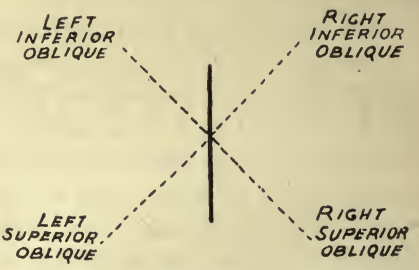

Fig. 37.—Diagram for the Investigation of Diplopia due to Paralysis of an Oblique Muscle of Either Eye. (After Werner.)

It is to be noted that *conjugate deviation of the eyes to one side* may be brought about by four kinds of lesion. Thus (1) an irritative lesion of the pons on the left side may cause deviation of both eyes to the left. (2) An irritative lesion of the right hemisphere may cause a similar deviation of both eyes to the left. (3) A destructive lesion in the left side of the pons may cause loss of deviation to the left and permit of a paralytic deviation to the right, through the action of the unopposed non-paralysed muscles. (4) A destructive lesion in the right hemisphere may cause a similar loss of conjugate movement to the left, and thus allow of a paralytic deviation to the right.

Paralysis of the Conjugate Lateral Movement of the Eyeballs results from a destructive lesion of the nucleus of the sixth nerve, or of the upper segment of the motor path leading from the cortex to that nucleus (Fig. 35). When the nucleus is destroyed, the external rectus is of course completely paralysed, but the opposite internal rectus can act in convergence, though not in conjugate movement of the two eyes. Loss of conjugate movement is a common transient symptom in hemiplegia, resulting from damage to the upper neurons, and is often accompanied by a similar deviation of the head away from the paralysed side.

The destructive lesion of the sixth nucleus may be softening, hæmorrhage or tumour. As the root-fibres of the seventh nerve wind round the sixth nucleus, the face is often involved at the same time.

Migraine with Recurrent Paralysis of the Third Nerve (called by Charcot *migraine ophthalmoplégique*, to distinguish it from the ordinary migraine with visual disturbance which he termed *migraine ophthalmique*). In the subject of this disease, an attack of severe unilateral headache, possibly accompanied by vomiting, is followed by ptosis on the affected side as well as by other evidences of impairment of the functions of the third nerve. After some days the paralysis lessens and may pass away, but in the course of time repeated attacks of headache and oculomotor paralysis occur, each paroxysm leaving more weakness behind it, until at last there is complete palsy of that nerve. The unilateral headaches still continue to recur. In those cases where a post-mortem examination has been made, there has been some structural change involving the trunk of the third nerve, such as tumour or exudation. The presumption is, therefore, that the earlier attacks are due to transient congestion or inflammation. The prognosis is serious so far as the nerve palsy is concerned, but there is not likely to be any danger to life. The iodides of iron and potassium may be tried, but the treatment is in the main purely symptomatic.

Ophthalmoplegia or Nuclear Ocular Palsy is in rare instances sudden (from a vascular lesion) or acute (from

inflammation), but much more commonly chronic and due to degeneration of the nuclei of the various ocular nerves. Ophthalmoplegia is described as *external*, *internal*, or *total*, according as the external or internal muscles or all the muscles of the eyeball are paralysed. Some of the palsies, however, are much more partial than even the expressions internal and external would suggest ; they may be limited to one muscle on each side (*e.g.*, the ciliary muscle), or to a single function of a muscle (*e.g.*, the contraction of the iris to light). Chronic ophthalmoplegia is specially apt to be met with in locomotor ataxy and in syphilitic subjects.

Reflex Iridoplegia is a common internal ocular palsy. While the pupil does not contract reflexly to light, it often contracts normally in looking at a near object, and when this is the case, it is spoken of as the 'Argyll-Robertson pupil.' It occurs in tabes dorsalis, general paralysis of the insane, and sometimes as an isolated phenomenon in syphilitic subjects.

Accommodation Iridoplegia : here the pupil fails to contract when the patient attempts to accommodate.

Cutaneous Iridoplegia is loss of reflex dilatation of the pupil on stimulation of the skin, for instance by pinching the skin of the neck. It occurs in tabes, etc.

Cycloplegia or paralysis of the ciliary muscle involves loss of accommodation so that an individual with normal refraction loses near vision, but this is restored by the use of a convex lens. It is one of the most common post-diphtheritic palsies, but also occurs in degenerative diseases of the nervous system.

Nystagmus consists in involuntary rhythmical clonic movements of the eyes, usually from side to side, but sometimes in other directions. It is seen in connection with visual defects (disease of the transparent media or of the fundus, albinism, etc.) ; in miners who work with the eyes turned in a direction involving constant strain of the ocular muscles ; and in certain diseases of the nervous system, including insular sclerosis, hereditary ataxy and cerebellar tumour.

Treatment of Ocular Palsies.—This consists mainly in removal of the cause. Thus in syphilitic cases, iodide of potassium and mercury; in rheumatic cases, alkalies and salicylates; and in chronic degenerative cases, arsenic, strychnine and iron, will be indicated. In acute neuritis, fomentations and leeches over the temple may be of service if applied in the earliest stages. Gowers speaks favourably of a blister applied behind the ear or at the occiput if the disease is at the base of the brain, and on the temple if it is in the orbit.

(5) Trigeminal Nerve.

This nerve has to do with sensation in the face, with the sense of taste, and with the movements of the masticatory muscles. It takes origin from the cervical region of the cord, as well as from the medulla and pons, and the lower part of its area of distribution is continuous with that of cervical spinal nerves.

The *motor functions* are easily investigated. The temporal and masseter muscles can be felt to contract when the mouth is closed. The lower border of the internal pterygoid can often be felt by pushing the finger up under the lower jaw near its angle. The external pterygoids pull the condyles forwards, so that if both are palsied, the lower jaw cannot be pushed forwards. If one only is paralysed, the jaw cannot be deviated towards the healthy side, and if the jaw is depressed it deviates to the affected side.

Spasm of the masticatory muscles (*trismus*) is an important symptom in tetanus, and occurs as an isolated phenomenon in hysteria.

The territory of the *sensory functions* extends as far laterally as the external auditory meatus, and includes the mucous membranes of the eyes, nose and mouth. Anæsthesia of these regions is often preceded and may be accompanied by symptoms of irritation, such as burning or neuralgic pains. The conjunctival reflex is lost; the mucous membrane of the nose is no longer irritated by ammonia, and the sense of smell is blunted or lost. Trophic changes may take place, the most important of which is *neuroparalytic*

ophthalmia. The cornea becomes cloudy, ulcerates, and may perforate, leading to destruction of the eye. This complication is seen chiefly when the nerve lesion is at or distal to the Gasserian ganglion. *Herpes zoster*, which is specially common in the territory of the first division of the nerve, is attributable to inflammation of the ganglion.

The Sense of Taste includes four qualities: bitter, sweet, salt and sour. The two former are best appreciated at the back, the two latter at the front of the tongue. The tests should be made with substances which have no odour, *e.g.*, quinine, sugar, salt and citric acid. The taste nerves are supplied to the anterior two-thirds of the tongue by the lingual branch of the third division of the fifth, and to the posterior third by the glosso-pharyngeal. The taste fibres, in passing to the brain, leave the lingual nerve to join the chorda tympani, and so reach the seventh nerve, in which they run as far as the geniculate ganglion. From this they are believed to pass by the great superficial petrosal and Vidian nerves to Meckel's ganglion, and thus to the second division of the fifth, in which they enter the brain. The taste fibres from the posterior third of the tongue and the palate enter the brain, not in the glosso-pharyngeal, but in the fifth nerve. It is supposed that they leave the glosso-pharyngeal at the petrosal ganglion by its tympanic branch (nerve of Jacobson), in which they reach the small superficial petrosal, and thus pass to the otic ganglion, and the third division of the fifth.

Division of the fifth nerve in man proximal to the Gasserian ganglion causes complete loss of taste (*ageusia*) on the corresponding side. Perversion of taste (*parageusia*) is occasionally present in hysteria and insanity, or as a sequel of influenza. Thus sweet articles may be regarded as bitter or metallic. Increased acuteness of taste (*hypergeusia*) may be met with under similar circumstances. *Subjective sensations of taste* may occur as hallucinations in insanity, as the aura of an epileptic fit, or as a result of disease of the nerves.

The fifth nerve may be damaged by lesions within the pons, *e.g.*, hæmorrhage, softening or tumour. It is often

involved at the base of the brain by gummatous and other forms of meningitis, tumours, caries of the bone or aneurysm. Primary neuritis is rare, apart from the inflammation of the ganglion which causes *herpes zoster*. Interstitial neuritis has been found in *hemiatrophy of the face*.

Diagnosis.—If there is merely pain from irritation of the nerve, the condition may be indistinguishable from neuralgia; but anæsthesia limited to the territory of the nerve, loss of taste on the same side as the pain, and loss of the motor functions of the nerve, are conclusive evidence that it is diseased. Loss of taste and loss of sensation on the same side of the face may result from disease at the posterior end of the internal capsule, and may also be observed as phenomena of hysteria; but in such cases there is hemianæsthesia, probably with impairment of the other special senses besides taste.

When the whole nerve is involved, the lesion is at the Gasserian ganglion, or at the base of the brain. In front of the Gasserian ganglion, disease may attack one or two divisions of the nerve. Thus, the first part may be affected by itself in the orbit or sphenoidal fissure. The second and third divisions may suffer together from disease of the sphenoid bone.

Disease at the side of the pons may involve the sixth or the third nerve with the whole of the fifth. Disease in the middle fossa of the base may affect these ocular nerves with the first division of the fifth. Disease in the pons may paralyse the fifth nerve on the side of the lesion, and the limbs of the opposite side (alternate hemiplegia).

Treatment.—The cause of the paralysis must be removed if possible, and the syphilitic, rheumatic or other constitutional state may require attention. If the disease is neuritis, a blister may be applied to the occiput, or behind the ear. Cocaine, and, if it fails, morphine, may be used to relieve severe pain. For the same symptom, phenacetin, gelsemium, or butyl chloral may be tried. If there is anæsthesia without pain, the skin should be stimulated by the faradic brush. The eyeball should be carefully watched, and if need be protected, so as to minimise the risk of ophthalmia.

(7) Facial Nerve.

The seventh nerve arises from a nucleus in the pons, and soon winds round the nucleus of the sixth nerve. Before leaving the pons it probably receives, by way of the posterior longitudinal bundle, fibres from the third nucleus for the orbicularis palpebrarum, and from the hypoglossal nucleus for the orbicularis oris. At the geniculate ganglion it receives the large superficial petrosal nerve, which contains taste fibres, but before it emerges from the Fallopian aqueduct these taste fibres leave it again by the chorda tympani.

Paralysis of the Facial Nerve (*Bell's paralysis*) may be due to a lesion (*e.g.*, hæmorrhage, softening, tumour) involving the nucleus or root-fibres in the pons. Such a lesion often paralyses the sixth nerve on the same side, and the limbs on the opposite side (alternate hemiplegia). At the base, the nerve may suffer (often along with the eighth nerve) from tumours, meningitis, etc. It often suffers within the temporal bone in children from otitis and bone disease ; there is almost always, in such cases, a history of discharge from the ear. Fracture of the skull, blows on the side of the head, surgical operations on the mastoid region, and pressure of the obstetrical forceps at birth may damage the nerve. Syphilis is an occasional cause of facial palsy. A very common cause in adults is inflammation resulting from cold—the so-called rheumatic neuritis. Occasionally facial neuritis is part of a multiple neuritis. Palsy may also result from nuclear degeneration. In rare cases facial palsy is bilateral (*facial diplegia*).

Of all kinds of facial palsy, that due to rheumatic inflammation is most common. The lesion is in the Fallopian canal, and as there is little room for swelling, the nerve fibres are compressed.

Symptoms.—When the palsy is complete, the affected side of the face is quite motionless. The difference between the two sides is specially marked in older subjects who have wrinkles on the face, as the wrinkles disappear on the affected side. The patient cannot close his eye, raise his brow, frown, smile or whistle. If he tries to shut his eye, the globe

rotates upwards. When he drinks, the liquid escapes at the side of the mouth. When he chews, food collects between the cheek and the teeth. It was formerly supposed, erroneously, that the palate is paralysed in facial palsy.

In severe facial neuritis the electrical reactions are altered, as in inflammation of other nerves.

If the facial nerve is diseased *between the geniculate ganglion and the origin of the chorda tympani*, not only are the above symptoms present, but taste is lost in the anterior part of the tongue. This loss is present in about half of the rheumatic cases. Another symptom of lesion in this region is increased sensitiveness of hearing (*hyperacusis*), especially with regard to musical notes of low pitch. This is due to paralysis of the stapedius, and consequent overaction of the tensor tympani.

In disease *between the pons and the geniculate ganglion*, taste escapes, but the auditory nerve often suffers, so that deafness is present on that side. The cause of the deafness and facial palsy often causes general head symptoms. If the auditory nerve should escape, there will be hyperacusis through palsy of the stapedius.

Disease of the *nerve or nucleus in the pons* is often associated with paralysis of the sixth nerve. Taste and hearing escape.

When exposure is the cause, the paralysis generally sets in within a couple of days, and reaches its height in a matter of hours or perhaps a day or two. In slight cases, recovery may take place within a fortnight, but the duration is very often several months, and recovery is often incomplete. Except in cases that are slight, and cases where the loss of power remains absolute, *contracture* sets in after some months in the weakened muscles.[1] It affects especially the zygomatici, and has the effect of restoring the lost naso-labial furrow. It is characterised by persistent tonic overaction (*secondary overaction*) of the weak muscles, but is accompanied by diminished capacity for strong voluntary action, and sometimes by irregular spontaneous twitchings. In old persons, this contracture restores the symmetry which was

[1] When muscles of the limbs are paralysed, it is their unparalysed opponents which undergo contracture.

lost through the palsy, but in the young it introduces a deformity.

Diagnosis.—It is important to bear in mind the contracture of late stages, so as not to suppose that the side which, when at rest, seems flatter than the other is the weak one. Disease of the nerve and nucleus (*infranuclear* and *nuclear* palsy respectively) must be distinguished from disease in the upper neurons of the motor path to the face muscles (*supranuclear* palsy). In disease above the nucleus, as in ordinary hemiplegia, the lower part of the face suffers more than the upper, and indeed the orbicularis palpebrarum and frontalis are seldom paralysed for more than a few days. In supranuclear (*cerebral*) facial palsy, emotional movement, as in smiling, is not impaired so much as voluntary movement, as in showing the teeth ; moreover, reflex movement is not lost, and the electrical reactions are not altered. In disease of the lower neurons (nuclear and infranuclear or *peripheral* palsy), the whole side of the face suffers alike, emotional movement and voluntary movement suffer equally, the reflexes are lost, and the electrical reactions are often altered.

The particular part of the facial nerve involved may be inferred from the affection of taste, etc., as indicated above.

Prognosis.—This is favourable in cases due to pressure by the forceps at birth, in multiple neuritis, and in syphilis. In the ordinary cases due to cold and to middle-ear disease, the most important element in prognosis is the electrical condition of the nerve. If after a fortnight it has lost none of its irritability, recovery may be expected in a few weeks ; if its irritability is quite lost, the duration will be several months at least. In a few instances the disease recurs.

Treatment.—Otitis, syphilis, or any other recognisable cause should be promptly dealt with by appropriate treatment. In rheumatic cases, treatment should be begun at the earliest possible moment. Hot fomentations should be applied at short intervals over the stylomastoid foramen, and a blister should be placed over the mastoid process a little further back. A purge and diaphoretic should be administered. In severe cases, it is well to galvanise the muscles several times a day. The positive pole may be

placed over the nerve or elsewhere, and the negative pole should be gently drawn along the various paralysed muscles.

An ingenious attempt has been made by Ballance and others to improve the condition of the face in incurable palsy of the seventh nerve, by making an anastomosis between the peripheral portion of the facial nerve and a neighbouring healthy nerve, such as the spinal accessory or the hypoglossal. In successful cases the tone is restored to some extent to the paralysed facial muscles, so that the symmetry of the face is regained to a corresponding degree. But there are serious disadvantages associated with the operation. The interference with the healthy spinal accessory nerve leads to more or less atrophy and weakness of the shoulder muscles. To move his face voluntarily the patient must raise his shoulder; and conversely, when he raises his shoulder, his face will move on that side. Moreover, emotional movement of the face remains absent. Atrophy of one half of the tongue, which results when the anastomosis is made with the hypoglossal, is probably less troublesome; but dysphagia has been noted as a result, and the operation is more difficult than when the spinal accessory is employed.

Facial Spasm (*mimic spasm, histrionic spasm, mimic cramp, convulsive tic*) is characterised by clonic spasm, and sometimes also by tonic spasm, in the muscles of one or both sides of the face. The orbicularis palpebrarum or the zygomatici commonly suffer first and most. The spasm may be almost limited to these, or may affect almost all the face.

The disease may be due to structural changes, either in the facial nerve (*e.g.*, from pressure by a tumour), or in the facial centre in the opposite hemisphere; or it may be idiopathic. The latter form occurs chiefly in the second half of life, is more common in women than in men, and is sometimes caused by mental strain and anxiety.

The spasm is aggravated by excitement and lessened by rest. There is no pain, but the mental distress caused by the affection may be considerable. The prognosis is not very favourable. Improvement or cure is likely to be temporary, and the patient may suffer all the rest of her life.

Treatment.—All possible causes of reflex irritation must be removed. Nerve tonics and sedatives (iron, quinine, strychnine, valerianate of zinc, Indian hemp, bromides) may be tried, but are rarely of any use. In one case which came under my observation, the lady appeared to derive much comfort from wearing dark spectacles.

Blepharospasm is bilateral spasm limited to the eyelids. It may be *tonic* or *clonic*. The former is seen in photophobia, and is an exaggeration of the conjunctival reflex. The latter occurs in hysteria and sometimes as a bad habit in children (*involuntary winking*, *nictitation*).

The **treatment** consists in removing the cause, immersing the face frequently in cold water, and administering tonics and sedatives.

(8) Auditory Nerve.

The auditory nerve consists of two parts which differ in function. The *vestibular* nerve, which is connected with the vestibule and semicircular canals, and is thus concerned in equilibration, enters the medulla at the inner aspect of the restiform body as the mesial root of the auditory nerve and passes to the dorsal auditory nucleus in the floor of the fourth ventricle. This nucleus is connected indirectly with the middle lobe of the cerebellum. The *cochlear* nerve, which is connected with the cochlea and the sense of hearing, is continued as the lateral root, external to the restiform body, and ends largely in the accessory auditory nucleus and the ganglion of the lateral root. Its central connections are with the posterior quadrigeminal and internal geniculate bodies, from which fibres are given off to the cortical auditory centre in the first temporal convolution.

Symptoms.—Among the symptoms which result from disease of the auditory nerve are loss of function (*deafness*) ; exaggerated sensitiveness to sounds (*hyperacusis*, *auditory hyperæsthesia*) ; irritation phenomena (*tinnitus aurium*, or *noises in the ears ; tinnitus capitis*, or *noises in the head*) ; and disturbance of equilibrium (*auditory vertigo*).

Etiology.—Disease of the labyrinth may be due to hæmorrhage, syphilis, or inflammation. The deafness which is

common in cerebro-spinal fever and often leads to deaf-mutism is probably due to inflammation of the labyrinth, since it is symmetrical and the facial nerves escape. Quinine and sodium salicylate cause deafness and tinnitus, probably by causing congestion of the labyrinth. The nerve may suffer at the base (often along with the facial nerve) through tumours, syphilitic and other forms of meningitis, caries, etc. Diseases of the middle ear and of the external meatus may cause various auditory symptoms.

TINNITUS AURIUM and TINNITUS CAPITIS are characterised by subjective noises in the ear and head respectively. The sounds vary greatly in different cases, and are often pulsating, keeping time with the arterial pulse. Elaborate subjective sensations of sound are usually referred by the patient to the external world, whereas simple sounds, including pulsating tinnitus, are generally referred to one or both ears or to the head. The symptom may result from any kind of ear disease, including, for instance, pressure of cerumen on the membrane. It is occasionally due to functional disturbance of the cortical auditory centre, as in the aura of an epileptic attack. Tinnitus is predisposed to by anæmia, neurasthenia, and gout.

Treatment.—The ear should be carefully examined, and any local disease treated. Any morbid constitutional state such as gout should also receive attention. Weir Mitchell's treatment may be indicated for neurasthenia. Bromide of potassium, with large doses of hyoscyamus, as a sedative, blistering over the mastoid, and in very chronic cases, sodium salicylate or quinine may also be tried.

MÉNIÈRE'S DISEASE[1] (*auditory*, *aural*, or *labyrinthine vertigo*) is characterised in typical cases by four symptoms: giddiness, sickness, tinnitus and deafness. The condition is apparently due to inflammation of the labyrinth, especially gouty or syphilitic inflammation, but very severe cases may be due to hæmorrhage. The name Ménière's disease is sometimes applied to cases with symptoms similar in kind to those mentioned, but slight in degree, and referable either to degenerative or other changes in the auditory nerve, or to

[1] Described by Ménière in 1861.

affections of the middle or external ear. These latter affections doubtless cause the symptoms by disturbing the intra-labyrinthine pressure. In well-marked cases, the symptoms occur in paroxysms at irregular intervals. An attack often sets in suddenly with tinnitus and a sense of reeling or actual reeling, which may after a time be followed by vomiting. Nystagmus may also be a symptom. Many cases recover, and many others improve, but sometimes the disease is progressive, and the vertigo ceases only when the nerve is so completely destroyed that hearing is abolished.

The **treatment** is similar to that recommended for tinnitus aurium. Intolerable vertigo has in some cases been relieved by operating on the labyrinth.

Endemic Paralytic Vertigo (*Gerlier's disease*), as seen in Switzerland, is similar to, if not identical with, *kubisagari*, which occurs in Northern Japan. Both affections are met with among people who live under the same roof with their cattle, and it has been supposed that the effluvium from the cattle is the cause. Both sexes and all ages are liable. The disease is characterised by attacks of ptosis, diplopia, dimness of vision, and paresis of various groups of muscles, and especially of those at the back of the neck, so that the head drops forwards. There is no loss of consciousness. The prognosis is favourable. Potassium bromide, potassium iodide and arsenic are reported to have proved beneficial.

(9) Glosso-pharyngeal Nerve.

This nerve is supposed to be sensory for the pharynx and tympanic cavity, and to be one of the motor nerves for the pharyngeal muscles. Though it contains taste fibres from the posterior third of the tongue and soft palate, these leave it at the petrosal ganglion and reach the brain in the fifth nerve. It is supposed that *nausea* depends upon the glosso-pharyngeal nerve.

The pharyngeal symptoms of bulbar paralysis are perhaps due to degeneration in the nuclei of the two ninth nerves. The nerve roots may be damaged, along with neighbouring

nerves, by meningitis, tumours, etc. A lesion distal to the petrosal ganglion may be expected to paralyse taste in the posterior third of the tongue.

(10) Vagus or Pneumogastric Nerve.

The root of the vagus is joined, soon after emerging from the jugular foramen, by the bulbar (internal or accessory) portion of the spinal accessory which rises from a continuation of the vagus nucleus in the medulla. This accessory portion has fine fibres, whereas the spinal (external) portion of the spinal accessory, which rises from the cervical region of the cord, has large fibres. The bulbar part of the spinal accessory contributes to the vagus the motor fibres for the larynx and soft palate, and also the cardio-inhibitory fibres. The vagus supplies the heart, larynx, lungs, pharynx, œsophagus, stomach and part of the intestine.

The nucleus of this nerve may be involved in bulbar paralysis. The nerve roots may be damaged by meningitis, tumours or aneurysms. The nerve may suffer in the neck from tumours, wounds, etc. The left *recurrent* branch is often compressed and paralysed by intrathoracic tumours, including aortic aneurysms, whilst the right recurrent nerve may be involved in apical pleuritic adhesions and in innominate aneurysm. The vagus may suffer with other nerves in multiple neuritis.

When the roots are affected, the twelfth nerve often suffers simultaneously, and a very characteristic group of symptoms consists of *unilateral paralysis of the tongue, soft palate and larynx.* As the superior laryngeal branch of the vagus is the sensory nerve of the larynx above the vocal cords, and the inferior or recurrent branch is the sensory nerve of the larynx below the cords, a lesion destroying the whole trunk or all the roots will cause *anæsthesia of the larynx* on that side. If both vagi are paralysed—*e.g.*, by diphtheritic neuritis—so that the *cardio-inhibitory nerves* are destroyed, there is acceleration and irregularity of the heart's action. Paralysis of the *pharyngeal branches* causes difficulty in swallowing. *Cheyne-Stokes breathing* is supposed to result from lowered activity of the vagus or respiratory centre.

Movements and Palsies of the Larynx—**Movements.**—The anterior ends of the vocal cords are fixed, being attached to the middle of the angle between the alæ of the thyroid cartilage; the posterior ends are attached to the mobile arytenoid cartilages. The glottis is opened and closed partly by the rotation of the arytenoid cartilages round their vertical axes, and partly by the movements of these cartilages away from and towards one another.

The *adductors* of the vocal cord include (1) the lateral crico-arytenoid which passes from the side of the cricoid cartilage upwards and backwards to the lateral or muscular process of the arytenoid cartilage. By drawing the muscular process forwards, this muscle moves the anterior or vocal process inwards and adducts the cord. (2) The thyro-arytenoid passes from the posterior surface of the thyroid cartilage to the vocal process of the arytenoid and to the cord itself. (3) The arytenoid muscle is unpaired and passes across the middle line, thus directly connecting the two arytenoid cartilages.

The *abductor* of the cord is the posterior crico-arytenoid which passes upwards and outwards from the posterior surface of the cricoid cartilage to the external or muscular process of the arytenoid. By pulling the muscular process backwards, it turns the vocal process outwards, and thus abducts the cord. The adjacent fibres of the lateral and posterior crico-arytenoids pull the arytenoid downwards and outwards, and thus tend to separate it from its fellow.

The cords are lengthened and made tense by the crico-thyroid muscles, which, passing from the front of the cricoid upwards and outwards to the thyroid, pull up the anterior part of the cricoid and thus depress its posterior part with the arytenoid cartilages.

The cords are shortened by the thyro-arytenoid muscles.

All the muscles named above, except the crico-thyroid, are supplied by the inferior or recurrent laryngeal nerve. The crico-thyroid is supplied by the superior laryngeal.

Paralysis.—This may result from bilateral disease of the cortical centre in the lowest part of the ascending frontal convolution, or of the motor path from the cortex to the

bulbar nuclei. The symptoms of such bilateral disease constitute *pseudo-bulbar paralysis*. Laryngeal paralysis is also a part of genuine bulbar or glosso-labio-laryngeal paralysis. The roots may be damaged by syphilis or aneurysm. The nerves may be involved in diphtheritic and other forms of multiple neuritis. Aneurysms, enlarged glands and other intrathoracic tumours may, as already stated, interfere with the nerves, and these may also become involved in pleuritic adhesions at the apices in chronic lung disease.

In other cases, the paralysis is functional and transient. This condition is met with in hysteria, anæmia and debility, but an exciting cause may be discoverable in catarrh of the larynx, over-use of the voice, etc.

In *hysterical* or *functional aphonia* the usual condition is paralysis of both adductors. The patient speaks in a whisper. The cords are far apart and cannot be brought together for phonation, though they are brought together in coughing. There is no dyspnœa or stridor. Adductor paralysis is rarely due to organic disease of the nerves or nerve centres.

In *paralysis of the abductors* (posterior crico-arytenoids), the cords are near one another, and cannot be separated except by the elasticity of the parts. In phonation and cough, they approximate still more closely. In course of time, the unopposed adductors undergo contracture, and tend to close the glottis almost completely. The voice is preserved, and the cough is normal, but respiration is seriously embarrassed. There is urgent dyspnœa, with loud inspiratory stridor, and marked upward and downward movement of the larynx. Expiration, however, is not interfered with. This condition may be due to disease of the nerve centres, nerves or muscles. In rare cases it is met with in hysteria.

Abductor paralysis is, however, commonly one-sided. Symptoms are then slight, and may be altogether absent. One of the most important causes of this condition is a lesion involving the recurrent nerve. Though this nerve contains both adductor and abductor fibres, it is the latter which suffer most readily from pressure. The affected cord

is near the middle line and cannot be moved outwards, whilst the other is freely movable.

Total paralysis of a vocal cord is characterised by the cadaveric position—namely, that which is assumed after death when all the muscles are relaxed. The position is one of slight abduction. The cord does not move inwards in phonation or outwards in deep inspiration. The voice is hoarse. The cough is brassy or imperfect. There is little or no stridor. This palsy may result from aneurysms and other intrathoracic tumours, goitre, etc.

In total paralysis of both cords, there is no voice, and no explosive cough. Both cords are motionless in the cadaveric position. The condition is rare, but may be produced by aneurysms of the aorta and innominate artery, cancer of the gullet, etc.

Prognosis of Laryngeal Palsies.—This depends mainly upon the cause of the paralysis. Functional paralysis may be expected to pass away under treatment. In paralysis due to multiple neuritis, a favourable ending may be hoped for, though the laryngeal element is a serious addition to the disease in its more usual aspect. Bilateral abductor paralysis is dangerous on account of the risk of suffocation. In other forms of laryngeal palsy, the latter is simply a detail, and often a small detail in the case.

Treatment.—The cause must be removed if possible, as in the case of a syphilitic lesion damaging the nerve roots. Very often, however, the cause cannot be removed. In cases due to hysteria, laryngeal catarrh, diphtheria, etc., strychnine should be administered, and blisters may be applied to the neck over the larynx. Bilateral abductor paralysis may necessitate laryngotomy or tracheotomy. In obstinate functional cases, electricity should be used, the poles being applied on either side of the neck. If this is insufficient, one pole should be placed within the larynx, and a momentary application of a strong faradic current will sometimes cause the patient to cry out at once. The voice may be thus restored, or a repetition of the treatment may be required. In many cases, general tonics are desirable.

Spasm of the larynx is met with in the laryngismus stridulus

of rickety children, and in the laryngeal crises of locomotor ataxy. It is possibly the cause of the initial cry in epileptic seizures.

Hunger and *vomiting* are supposed to be related to the vagus, which gives both sensory and motor fibres to the stomach. It is possible that intracranial disease causes vomiting by irritating the roots or the nucleus of this nerve.

(11) Spinal Accessory.

The *accessory* or bulbar portion of this nerve joins the vagus and has been already considered.

The *spinal* portion rises from the cervical region of the cord, enters the head and leaves it again, and is distributed to the sterno-mastoid and the trapezius. It may suffer in or near the cranium from meningitis, tumours or aneurysms. The cells of origin may be involved in progressive muscular atrophy. The roots may be damaged in caries of the cervical spine. After leaving the skull the nerve may be paralysed by direct injury, spinal caries, abscesses connected with the cervical glands, etc.

The result in any case is weakness and wasting of the sterno-mastoid and the trapezius. Torticollis does not result, but there is impaired power of rotating the head to the opposite side, and of elevating the shoulder on the side of the lesion. If the nerve is injured after it perforates the sterno-mastoid, that muscle will of course escape.

Torticollis is described under Functional Nervous Diseases.

Treatment.—The cause must be removed if possible—*e.g.*, pressure in cervical caries. Electrical stimulation of the paralysed muscles should be practised. In progressive muscular atrophy, strychnine should be injected hypodermically in small doses once a day.

(12) Hypoglossal Nerve.

This is a purely motor nerve and is distributed to the muscles of the tongue and those connected with the hyoid bone. Its nucleus in the medulla sends some fibres by the posterior longitudinal bundle to issue in the facial nerve for the supply of the orbicularis oris.

Paralysis.—The nucleus degenerates in some cases of bulbar paralysis. The nerve roots are damaged by aneurysms and other tumours, meningitis, caries, etc.

The most obvious result is weakness and wasting of the corresponding half of the tongue. The tongue, when protruded by the unaffected genioglossus muscle, deviates towards the affected side. The mucous membrane of the wasted half is thrown into folds, and fibrillary twitchings may be observable.

When unilateral paralysis is due to disease of the roots, it is sometimes accompanied by unilateral paralysis of the palate, laryngeal muscles, sterno-mastoid and part of the trapezius, in consequence of simultaneous involvement of the spinal accessory nerve.

When both sides of the tongue are paralysed, the tongue lies motionless in the mouth and cannot be protruded. Articulation and mastication are impaired in bilateral, but scarcely at all in unilateral disease.

If the weakness is due to disease above the nucleus, wasting is absent. Weakness of one side of the tongue is common in ordinary hemiplegia.

The **prognosis** is usually unfavourable.

Treatment consists in the removal of the cause, so far as this is possible; the administration of tonics internally; and the application of electricity to the tongue.

Spasm of the tongue occurs in some cases of stammering, chorea and epilepsy. It may also be due to irritation of the nerve by disease involving the roots at the surface of the medulla—for instance, gummatous meningitis.

vi. Diseases of the Spinal Nerves.

Phrenic Nerve.

The phrenic nerve is derived from the fourth cervical, and in part from either the third or the fifth cervical. It is the motor nerve of the diaphragm, and it also gives branches to the pericardium, pleura and peritoneum. Its function may be impaired through disease of the spinal cord, or its roots may be injured by meningitis or disease of the spine.

The nerve is sometimes involved in multiple neuritis. Phrenic neuritis may follow exposure to cold. The nerve may be injured by wounds in the neck and by aneurysms and other tumours in the chest.

Hiccough (*singultus*) is due to clonic spasm of the diaphragm, combined, probably, with insufficient opening of the glottis. It may be neurotic, or due to some inflammatory affection of a serous membrane, or to some other agency which may irritate the diaphragm or phrenic nerve directly, or in a reflex manner—*e.g.*, by way of the vagus.

Symptoms.—The paralysis of the diaphragm which results from disease of the phrenic nerve is difficult to detect unless both nerves are affected. When the paralysis is bilateral, respiration is of the costal type. The epigastric region, instead of being protruded, is drawn in with each inspiration, and it becomes more prominent with expiration. Breathing is not much interfered with as long as the patient is at rest, but dyspnœa may quickly set in on exertion. The most serious result of diaphragmatic palsy is that it adds much to the dangers attendant upon any inflammatory condition in the respiratory system.

Treatment.—When the nerve is inflamed, a blister should be applied over it in the neck as early as possible. In later stages, hypodermic injections of strychnine may be beneficial. The patient should be specially guarded against the risks of bronchitis, pneumonia, etc.

Hiccough is sometimes very obstinate. In slight cases it may be arrested by holding the breath, or by breathing very deeply and slowly. In more severe cases, the sucking of ice, the application of a mustard plaster or of the ether spray to the epigastrium, strong traction on the tongue, washing out the stomach by the tube, galvanisation of the phrenic nerve, the inhalation of chloroform or of amyl nitrite, and the hypodermic injection of morphine or of apomorphine, are remedies which may be borne in mind and tried as required.

Brachial Plexus.

The brachial plexus is formed from the anterior primary divisions of the four lowest cervical and first dorsal nerves.

The plexus may be paralysed in whole or in part—*e.g.*, by growths involving the nerve roots ; by a sudden strain on the arm stretching or tearing the nerves ; by the pressure of the dislocated head of the humerus ; by fractures of neighbouring bones ; or by extension of inflammation from a nerve below, or a nerve root above the plexus. The interference with movement and sensation caused by a dislocation may be very transient if the dislocation is speedily reduced. But if the reduction is long postponed, or if the nerves have been lacerated, the muscles will become the seat of wasting as well as weakness, sensation may be seriously disturbed, and trophic changes in the skin may ensue.

In cases of unilateral injury to the brachial plexus by stretching, smallness of the pupil on the affected side suggests detachment of the roots from the spinal cord rather than rupture of the trunks distal to the junction of the anterior and posterior roots.

The *Duchenne-Erb paralysis* is a partial paralysis of the brachial plexus due to lesion of the fifth and sixth cervical roots. The palsy involves the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the supraspinatus, infraspinatus and supinator brevis. This paralysis is not uncommonly met with in the newborn child as a result of a lesion occurring during the process of birth, whence Duchenne called it *birth paralysis*. It appears from Kennedy's observations that the seat of lesion is the junction of the anterior divisions of the fifth and sixth cervical nerves. If an operation is performed for non-recovery, what is generally found is a cicatricial mass resulting from complete rupture. The lesion is due to overstretching of the fifth and sixth nerves by forcible depression of the shoulder, combined with bending of the head to the opposite side, and probably rotation in one direction or another. It does not depend upon any particular presentation of the child. Some of these cases recover perfectly, but if after the lapse of two months there is little or no evidence of the return of power, the question of operation should be considered, as this has yielded very encouraging results. Kennedy's operation consists in excision of the cicatricial mass referred to by

dividing the fifth and sixth nerves (both above the lesion), and the suprascapular nerve, the branch to the outer cord, and the branch to the posterior cord of the plexus (all three below the lesion), and then joining the two central ends to the three peripheral ends by catgut suture.

Klumpke's paralysis is a partial palsy of the plexus due to lesion of the eighth cervical and first dorsal nerves. The muscles involved are the intrinsic muscles of the hand and the flexors of the fingers and wrists.

Brachial Neuritis.

Brachial neuritis sometimes occurs as a primary peri-neuritis, involving the nerves of the brachial plexus in the same way as sciatica involves the great sciatic nerve. Indeed, the condition has been termed 'sciatica of the arm.' A gouty tendency and advanced life are important etiological factors.

Symptoms.—Pain, often severe, paroxysmal, and radiating, is the first and most distressing symptom. The skin may be hyperæsthetic. The motor nerves are not as a rule much affected. The course is often tedious, and recovery may be incomplete.

In the **treamtent** of this condition all movement that causes pain must be avoided. Fomentations should be applied in the early stage. Cocaine should be injected hypodermically. Later on gentle friction may be employed to get rid of stiffness.

Posterior Thoracic Nerve.

The posterior thoracic or long thoracic nerve may be injured by direct pressure in the posterior triangle of the neck through carrying loads on the shoulder ; or by constant contraction of the scalenus medius, as in plasterers who have the arm raised at work, this muscle being perforated by two of the roots of the nerve. According to Gowers, this paralysis is nine times as common in men as in women. It is seen chiefly in the first half of adult life, in muscular workers, and on the right side of the body. Several of these facts are related to occupation.

Symptoms.—The characteristic result of disease of this

nerve is paralysis of the serratus magnus. With the arm at rest the lower angle of the scapula is tilted inwards by the rhomboids. When the patient pushes in a forward direction, the posterior edge of the scapula projects to an abnormal extent from the chest, and a deep groove is produced along the inner border of the bone. At the same time, the lower angle is tilted inwards and upwards. Elevation of the arm above the shoulder is weakened but not lost. In cases due to neuritis there may be severe pains about the shoulder.

Treatment.—The arm on the affected side should be kept in a sling so as to raise the shoulder. In the early stages, a blister may be applied over the scalenus. Galvanism should be used to preserve the nutrition of the muscle.

Circumflex Nerve.

The circumflex nerve is readily injured by dislocation of the shoulder, or by the individual falling upon the shoulder, or by the pressure of a crutch (*crutch palsy*).

Symptoms.—The resulting palsy of the deltoid is easily recognised by the impaired power of abduction of the upper limb. Wasting and electrical changes ensue in severe cases. The loss of function of the teres minor is not very easily detected. Sensation may be impaired in the skin over the deltoid, and trophic changes and adhesions in the shoulder-joint sometimes develop.

Musculo-spiral Nerve.

The musculo-spiral nerve suffers from blows, fractures, the pressure of a crutch (this being the most common form of crutch palsy), and pressure in sleep. The individual may lie upon his arm, pressing it under him, or he may fall asleep with the limb resting on the edge of the bed or the back of a chair. These *sleep palsies* are particularly frequent in, though not confined to, persons who have taken alcohol in excess. Individuals who have fallen asleep in a natural way are usually awakened by the tingling and anæsthesia which are soon induced by continuous pressure on a

nerve, whereas alcoholic sleep may be undisturbed for hours by any such cause, and a serious neuritis is set up.

Symptoms.—Complete paralysis involves the extensors of the elbow, wrist and digits, and the supinators. But the nerve is usually affected after giving off its branches to the triceps, so that the latter muscle escapes. If the lesion is as low as the division into radial and posterior interosseous, the supinator longus and extensor carpi radialis longior escape. Wrist-drop is a marked feature, and flexion of the wrist and fingers is impaired owing to the lack of purchase which results. Sensation is usually much less involved than movement.

Bilateral wrist-drop with escape of the supinators is a common result of lead poisoning, and is due to neuritis. Wrist-drop, however, occurs in other kinds of neuritis, including the alcoholic variety.

Median Nerve.

The median nerve sometimes suffers in sleep palsy, and in fractures of the forearm bones. Disease of the nerve causes loss of pronation beyond the mid-position. The flexor carpi radialis and the flexor longus pollicis are paralysed. The loss of function of the latter interferes with opposition of the thumb to the finger-tips, with the holding of a pen, and with the picking up of small articles. Flexion of the proximal interphalangeal joints is lost ; and flexion of the distal joints of the index and middle fingers is also lost. Sensation is impaired or lost on the palmar aspect of three and a half digits, counting from the radial side, and on the corresponding portion of the palm of the hand.

Ulnar Nerve.

The ulnar nerve may be damaged by fractures and dislocations at the elbow, or by prolonged stretching over the flexed elbow in patients who are imperfectly sensible through fever. When the nerve is diseased, the ulnar flexor of the wrist is paralysed, so that in flexion the hand is drawn to the radial side. Flexion of the distal joints of the little and

ring fingers is lost, but flexion of the proximal interphalangeal joints is preserved. The paralysis of the interossei and two inner lumbricales causes loss of flexion of the metacarpophalangeal and loss of extension of the interphalangeal joints, the loss being greatest in the two inner digits. Adduction and abduction of the fingers are also lost. The hypothenar eminence becomes wasted. Sensation is more or less lost in the little finger and ulnar half of the ring-finger, on both palmar and dorsal aspects, and in the corresponding portions of the hand. The unopposed long muscles tend to evolve a claw-hand (*main en griffe*).

Lumbar Plexus.

The lumbar plexus is formed from the anterior primary divisions of the first three lumbar nerves and half of the fourth. It is sometimes injured by growths in the lumbar glands and by a psoas abscess. Inflammation may extend to it by the lumbo-sacral cord or may occur spontaneously. Its roots may suffer in disease of the vertebræ and in meningitis. The plexus supplies the skin of the lower part of the abdomen, the front of the thigh, and the inner aspect of the leg and foot; it also supplies the cremaster muscle, the flexors of the hip, the adductors of the thigh, and the extensors of the knee. Paralysis of the plexus will lead to corresponding defects of sensation and movement.

Anterior Crural Nerve.

The anterior crural nerve may be injured by fractures and dislocations at the hip, by wounds of the groin, and in parturition.

Symptoms.—Disease of this nerve causes paralysis and wasting of the extensors of the knee, with loss of the knee-jerk. If secondary contracture of the flexors takes place in both limbs, walking and standing are impossible. Rising from the kneeling posture is impossible without the aid of the hands. Moreover, there is anæsthesia of the inner aspect of the foot and leg, and of the front and inner aspect of the greater part of the thigh.

Obturator Nerve.

This nerve may be damaged in parturition or by a tumour. The result is paralysis and wasting of the adductors of the thigh, so that the crossing of one leg over the other is prevented, and riding is difficult.

Sacral Plexus.

This plexus is derived from the lumbo-sacral cord (half of the fourth with the fifth lumbar nerve) and the first four sacral nerves. It supplies the extensors of the hip, the flexors of the knee and all the muscles which move the foot. It also supplies the skin on the back of the hip and thigh, the outer side and back of the leg, and most of the foot. The plexus may be injured by compression in labour, by pelvic inflammation and by growths. It is often the seat of inflammation, and this has generally ascended to it from the sciatic nerve.

The symptoms will be in accordance with the distribution of the plexus as already indicated.

Superior Gluteal Nerve.

This nerve is rarely paralysed alone. It supplies the gluteus medius and minimus, so that disease causes loss of abduction of the thigh.

Great Sciatic Nerve.

The great sciatic nerve may be injured by wounds, dislocations of the hip and fractures of the femur. Paralysis is seldom complete, but a lesion high in the course of the nerve paralyses the hamstring muscles and all the muscles below the knee. Walking is still possible through the activity of the hip muscles. *Sciatica* will be described separately.

Small Sciatic Nerve.

The small sciatic nerve is rarely paralysed alone. Disease causes anæsthesia of the thigh and upper part of the calf on their posterior aspect.

External Popliteal Nerve.

The external popliteal or peroneal nerve, from its superficial course and proximity to the bone, readily suffers from traumatic causes, and is also liable to spontaneous neuritis.

Symptoms.—Paralysis causes ankle-drop, with dragging of the toes and a high step in walking. There is loss of flexion of the ankle and of extension of the first phalanges of the toes. The muscles in the front of the leg undergo wasting. Contracture of the unopposed calf muscles may lead to *pes equinus*. There is anæsthesia of the dorsum of the foot and of the outer aspect of the leg.

Internal Popliteal Nerve.

This nerve supplies the popliteus, calf muscles, long flexors of the toes and muscles of the sole.

Symptoms.—Paralysis causes loss of extension of the ankle, and loss of flexion of the toes. The patient is unable to stand on tiptoe. *Talipes calcaneus* and clawfoot may develop.

Sciatica.

Sciatica is a painful affection of the great sciatic nerve,[1] and may be either *primary* or *secondary*.

Etiology.—Males are several times as liable as females. The disease does not occur in children. It is most common between thirty and sixty. Gout, and exposure to cold and wet, are important causes. Sciatica often exists in association with lumbago, both being due to rheumatic inflammation of fibrous tissue. Pressure on the nerve is another cause, as in miners who habitually work while lying on their side, and are thus liable at the same time to have their clothes wet.

Secondary sciatica may be caused by pelvic tumours and inflammation, injury during labour, and disease of the hip-joint. The symptoms are doubtless partly due to pressure, and therefore neuralgic in their nature, but they may be due in great measure to inflammation excited by that pressure or extending to the nerve from the original lesion.

[1] 'The sinew that shrank' (Gen. xxxii. 32) is the sciatic nerve.

Morbid Anatomy.—There is neuritis which involves chiefly the nerve-sheath, but may extend into the interstitial tissue and may even damage the nerve fibres. In the early stages, the sheath is swollen and red. The changes are most marked at the sciatic notch and at the middle of the thigh.

Symptoms.—The most important symptom is pain in the great sciatic nerve and often in its branches. It varies in character and is frequently severe. Tenderness is usually present in the same parts, so that the area involved in a severe case extends from the sciatic notch to the outer border of the foot. Tenderness (due to involvement of the *nervi nervorum*) is specially marked at the lower border of the gluteus, the middle of the thigh, the middle of the popliteal space, below the head of the fibula, and behind the external malleolus. The pain is increased by any posture, movement, or other agency which increases the tension on the nerve. Occasionally there is defect of sensation at the back of the thigh, indicating involvement of the small sciatic. Muscular wasting is usually trifling or absent.

In *secondary* sciatica, there is seldom so much tenderness in the nerve trunk as in the primary disease.

Diagnosis.—Sciatica must be clearly distinguished from pain radiating along, or neuralgia of, the sciatic nerve. True sciatica is almost invariably confined to one side, and sciatic pains on both sides should suggest some cause in the pelvis, or even more in the spine, spinal membranes, cauda equina or spinal cord. Persistent tenderness points to neuritis, either primary or secondary. Neuralgic pain being excluded, it is necessary to ascertain whether the nerve affection is primary or secondary. The hip-joint, sacro-iliac joint, spine, and, in doubtful cases, the pelvic organs, should be carefully examined.

Prognosis.—In mild cases, recovery may take place within a few weeks if the patient rests from an early stage. In more severe cases, the duration may be many months. Relapses may occur, and as recovery is taking place in the one limb, the disease may attack the other.

Treatment.—Rest is essential. At the outset a mercurial purgative and a diaphoretic should be given. The local

application of an icebag should be tried at once. Repeated small blisters are sometimes of service, each being applied in succession to the varying seats of maximum pain. Severe pain should be treated by cocaine hydrochloride injected hypodermically once or twice a day, but only for the first few days; hot fomentations may also be applied. In severe cases a needle may be inserted into the most painful part of the nerve and left there for a quarter of an hour; or distilled water, or solutions of morphine, cocaine, or sodium salicylate (5 grains in 1 drachm) may be injected into the nerve, one each day in rotation. The galvanic current may be tried in the later stages, the sedative positive pole being placed over the seat of pain. Occasionally the nerve has been stretched with satisfactory results, but this must not be attempted till the acute stage has passed away. The stretching is done under chloroform, either by extending the knee and flexing the hip as strongly as possible, or by exposing the nerve and pulling on it strongly with the hand.

DISEASES OF THE SPINAL CORD.

i. Introduction.

The spinal cord contains centres of reflex action and centres which control the nutrition of tissues. It also contains numerous tracts of nerve fibres, some of which convey impulses from one part of the cord to another, whilst others convey impulses between the cord and the brain in either direction. Some of these TRACTS must be mentioned here (Fig. 38).

(1) The *crossed* or *lateral pyramidal tract* is derived from the motor area of the cortex on the opposite side, and crosses the middle line in the pyramids of the medulla. It runs in the posterior part of the lateral column of the cord, and is in part separated from the surface by the lateral cerebellar tract. It constitutes the upper segment of the motor path, and consists of the axis-cylinder processes of cells in the motor area.

(2) A minority of the fibres of the pyramidal tract coming

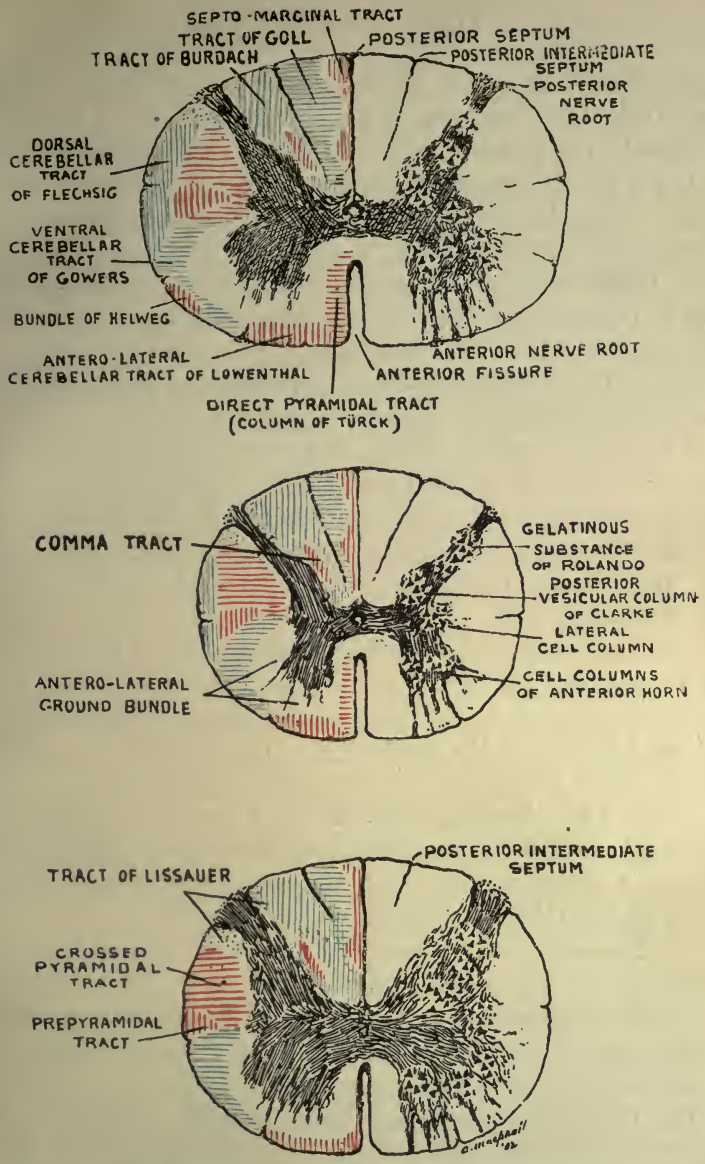

FIG. 38.—DIAGRAMMATIC SECTIONS OF THE SPINAL CORD IN THE CERVICAL, MID-DORSAL, AND LUMBAR REGIONS.

The relative proportions of grey and white matter at the different levels are shown. On the one side the groups of cells are indicated, and on the other the principal fibre-tracts. Ascending tracts are coloured blue, and descending tracts red. Each tract is shaded uniformly in all the sections, so that its extent and its relations to other tracts can be readily followed. It is to be noted that the septo-marginal and comma tracts, though descending and therefore coloured red, are afferent in function.

from the motor cortex do not cross the middle line at the pyramids, but run down in the anterior column of the cord alongside the anterior median fissure. These fibres constitute the *direct* or *uncrossed pyramidal tract* or *column of Türck*. These fibres doubtless cross gradually, lower down, by way of the anterior commissure.

A third group of fibres, not recognisable as a separate tract, pass at the level of the pyramids into the lateral pyramidal tract of their own side.

These three classes of fibres belonging to the pyramidal system are all to be regarded as commencing in the cells of the motor area of the cortex, and as ending by breaking up into arborisations around motor cells of the anterior cornua of the spinal cord.[1] The arborisations are the lower ends of the upper neurons of the motor path. The motor cells of the anterior horns are the upper ends of the lower neurons.

(3) The *antero-lateral ascending tract of Gowers* lies in front of the crossed pyramidal and lateral cerebellar tracts, and passes by way of the medulla, pons and superior peduncle to the middle lobe of the cerebellum. It probably takes origin from cells in the posterior horn of the opposite side.

(4) The *direct lateral cerebellar tract* lies at the surface of the cord in the lateral region. It consists of the axons of the cells of Clarke's column, and passes up by way of the restiform body to the middle lobe of the cerebellum.

(5) *Lissauer's tract* is situated at the surface of the cord opposite the tip of the posterior horn. It consists of fine ascending fibres which have entered by the posterior roots.

Between the posterior median fissure and the posterior horn there are two tracts.

(6) The *postero-external column*, *column of Burdach*, or *posterior root-zone* contains fibres from the posterior roots, of which some pass through it into the posterior horn, and others, passing upwards, incline towards, or into, the postero-median column. The axons of this column have their trophic cells in the posterior root ganglia, and many of them terminate in the nucleus cuneatus at the posterior

[1] See footnote, p. 621.

aspect of the medulla. In the middle of the anterior portion of this column is the *comma tract* of descending branches of posterior root fibres (see p. 625).

(7) The *postero-median column* or *column of Goll* consists largely of uncrossed fibres from the posterior roots, which, as they pass upwards, gradually incline through the postero-external column to enter the postero-median. Their cells are in the posterior root ganglia, and their axons end in the nucleus gracilis of the medulla.

Secondary Degeneration, Ascending and Descending.—Just as a peripheral nerve fibre degenerates after being cut off from its trophic cell, so do axons running in the central nervous system degenerate when deprived of the trophic influence of their cells of origin. Such deprivation may be due to severance of the axon or to disease of the cell. In nerve tracts which conduct upwards, secondary degeneration is found above the lesion (*ascending degeneration*), and in tracts which conduct downwards, degeneration is found below the lesion (*descending degeneration*). The direction in which a tract degenerates is thus an evidence of the direction in which it conducts impulses. A lesion involving the whole thickness of the spinal cord will therefore be followed by secondary degenerations both above and below the lesion. A hæmorrhage which tears up the internal capsule on one side of the brain is followed by secondary degeneration of the pyramidal tract, in its whole length below the seat of lesion.

System Diseases.—While certain diseases of the cord, and especially inflammation, attack its substance in a more or less random fashion, there is an extensive group of affections characterised by primary degeneration of one or more sets of neurons which possess a common function. Thus in locomotor ataxy, there is degeneration of the afferent neurons of the posterior roots, while in progressive muscular atrophy, the lower motor neurons, and in primary lateral sclerosis, the upper motor neurons degenerate. Ataxic paraplegia and Friedreich's hereditary ataxy are examples of *combined sclerosis*, both being characterised by degeneration of more than one tract of fibres on each side. Diseases in which

TABLE SHOWING APPROXIMATE RELATION TO THE SPINAL NERVE ROOTS OF CERTAIN MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE CORD.

	Motor.	*Cutaneous.*	*Reflex.*
C. 1.	Small rotators of head.		
2.	Depressors of hyoid bone. Sterno-mastoid.		Sudden inspiration on sudden pressure beneath lower costal margin.
3.	Scaleni. Levator anguli scapulæ. Trapezius.	Occiput and neck. Top of shoulder.	
4.	Diaphragm.		
5.	Shoulder abductors.	Outer aspect of upper limb.	
6.	Elbow flexors. Serratus. Shoulder adductors.		Dilatation of pupil.
7.	Extensors of wrist and digits. Elbow extensors. Latissimus.	Digits.	Scapular.
8.	Small muscles of hand. Flexors of wrist and digits. Pectorales.	Inner aspect of upper limb.	
D. 1.	Intercostals. Rectus et obliqui abdominis.	Skin of chest and abdomen. Ensiform region (6-7). Umbilicus (9-10).	
2.			
3.			Epigastric.
4.			
5.			
6.			
7.			Abdominal.
8.			
9.			
10.			
11.			
12.			
L. 1.	Cremaster.	Hip.	
2.	Hip flexors.	Anterior surface of thigh.	Cremasteric.
3.	Adductors and knee extensors.	Knee, except at back.	Knee-jerk.
4.		Inner aspect of leg.	Gluteal.
5.	Abductors.		
S. 1.	Hip extensors and knee flexors.	Root of penis dorsally.	Plantar.
2.		Back of limb and outer border of foot.	Ankle clonus.
3.	Perineal muscles.	Perineum, lower part of scrotum, and most of penis.	
4.			
5.			

Splenius. Complexus. — Erector spinæ. — Foot muscles.

particular systems of neurons are involved are known as *system diseases of the cord*, and are distinguishable from *focal*, *disseminated* and *diffuse* lesions. The last three terms, however, will be more appropriately considered in connection with the subject of myelitis (p. 686).

A TOTAL TRANSVERSE LESION of the cord abolishes sensation and voluntary movement so far as the parts connected with the cord below the lesion are concerned. By noting in the first place how far up the limbs and trunk the motor and sensory loss extends, and by studying in the second place the symptoms of irritation of the lowest healthy nerve fibres (cutaneous hyperæsthesia, girdle sensation, radiating pains), we can estimate the level of the upper limit of the lesion. This process is aided by a TABLE (see page 678) showing which regions of the cord are connected with particular muscles, cutaneous areas, and reflex functions. A total transverse lesion also causes loss of the deep reflexes below the lesion, with muscular wasting, loss of muscular excitability to faradism, and loss of tone of the sphincters. The only evidence of independent activity remaining in the isolated part of the cord is the occasional presence of certain skin reflexes in slight degree. Gowers considered that these phenomena were indicative of disease existing in, or spreading to the lumbo-sacral enlargement, but it is now known that they may occur in the absence of any recognisable change

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According to Head, the sensory fibres of the viscera probably originate as follows :

Heart.—Second to eighth dorsal.

Lungs.—Third and fourth cervical ; third to ninth dorsal.

Stomach.—Sixth to tenth dorsal (cardiac end, sixth and seventh dorsal ; pyloric end, tenth dorsal).

Intestines except Rectum.—Tenth to twelfth dorsal.

Rectum.—Second to fourth sacral.

Liver and Gall-bladder.—Seventh to tenth dorsal.

Kidney and Ureter.—Tenth dorsal to first lumbar.

Bladder Muscle.—Eleventh dorsal to first lumbar.

Bladder (*Mucosa and Neck*).—Second to fourth sacral.

Testis and Ovary.—Tenth dorsal.

Uterus.—Tenth dorsal to first lumbar.

Os Uteri.—Second to fourth sacral.

in the anterior horn cells, the anterior roots, or the peripheral nerves of the paralysed region. The loss of the knee-jerk, therefore, in complete transverse lesions of the cord must be a result of isolation from the higher centres.

A Unilateral Transverse Lesion causes loss of power on the same side below the lesion. From a little *below the lesion* downwards, there is hyperæsthesia on the same side and anæsthesia on the opposite side. The hyperæsthesia is due to the irritation of nerve fibres in the sensory path by the changes taking place in the healthy cord at the upper limit of the destructive lesion. When a sensory nerve tract is irritated the sensation induced is referred to the peripheral distribution of that tract, which in the present case is the part of the body below the lesion and on the same side as the lesion. As the sensory path crosses over to the other side of the cord very soon after entering it, the zone of anæsthesia below the lesion which results from destruction of the path is of very limited vertical extent. The anæsthesia on the opposite side of the body is due to destruction of the sensory path which has crossed from the opposite side to the side of lesion below the lesion. Muscular sensibility, however, differs from the other kinds of sensation, and if affected at all suffers on the same side as movement. This is because the afferent path from the muscles passes up in the posterior columns of the same side, and does not cross in the cord. Reflex action is ultimately increased on the side of lesion. *At the level of lesion*, and on the side of lesion, there may be a zone of anæsthesia from damage to entering nerve fibres; and immediately above this, there may be hyperæsthesia from irritation of nerve fibres. The arrangement of symptoms is shown by the following table from Gowers :

Zone of Cutaneous Hyperæsthesia. ,, ,, *Anæsthesia.*	*Lesion.*	
Motor palsy.		Muscular power normal.
Cutaneous hyperæsthesia.		Cutaneous anæsthesia.
Muscular sense impaired.		Muscular sense normal.
Reflex action first lessened, then increased.		Reflex action normal.
Temperature raised.		Temperature same as above lesion.

The combination of symptoms arising from a unilateral transverse lesion of the cord has been called *Brown-Séquard's paralysis*.

ii. Spinal Meningitis.

Spinal meningitis may be *acute* or *chronic*, *diffuse* or *localised*. It may involve the dura mater (*pachymeningitis*) or the pia mater and arachnoid (*leptomeningitis*). Chronic inflammation may be confined to one membrane, but acute inflammation, though beginning in one, is apt to spread to the others. These inflammations are classified as *external* or *internal*, according as they begin outside or inside the dura mater. Internal meningitis may be acute or chronic, the former beginning in the soft membranes, the latter either in the pia-arachnoid or on the inner surface of the dura. External meningitis also may be acute or chronic.

Acute Internal Meningitis.

In this disease the inflammation sometimes spreads to the inner aspect of the dura, and frequently to the spinal cord (*meningo-myelitis*). It may be part of a cerebro-spinal meningitis, due to the organism of the epidemic disease, or to the tubercle bacillus; both of these varieties have been described in Section I. It also occurs in pneumonia and other infectious diseases. It may appear to be due to cold or to an injury of the spine.

Among the organisms which may be the immediate cause of acute leptomeningitis are the *Bacillus tuberculosis*, the *Diplococcus intracellularis meningitidis*, the *Pneumococcus*, the *Streptococcus*, the *Staphylococci*, and the organisms of numerous acute infections such as diphtheria, enteric, influenza, and gonorrhœa.

Morbid Anatomy.—The inflammation is usually widespread. At first there is congestion of the membranes, but after a time the meshes of the pia-arachnoid become filled with a greyish-yellow or more distinctly purulent exudation. This surrounds the nerve roots, and may fill up the whole space between pia and dura. In the tubercular form, however, the exudation is scanty, and grey tubercles may be recognised.

Symptoms.—The onset is acute, with a rigor, fever, and pain in the back. The irritation of the meninges accounts for the continuous pain along the spine, which is often aggravated by movement, by pressure, and by the local application of a hot sponge. The irritation of the sensory nerve roots causes paroxysms of pain which radiates along the nerves, around the trunk and into the limbs. Spasm, partly reflex and partly due to irritation of motor roots, manifests itself in rigidity of the muscles, especially of the back. Other symptoms are cutaneous hyperæsthesia, constipation, retention of urine, Kernig's sign (p. 78), and the *tache spinale* (the excessive and prolonged dilatation of cutaneous vessels which follows a stroke with the finger-nail, pointing to interference with vasomotor nerves). Later on there is a tendency to loss of motion, of sensation, and of reflex action, showing that destruction has succeeded to irritation of nerve structures.

Diagnosis.—The acute onset of fever, with pain in the back aggravated by movement, muscular rigidity, and cutaneous hyperæsthesia, is quite characteristic. In *myelitis*, unless meningitis be also present, there is little or no pain in the back, and paralysis occurs early and is much more marked than spasm. In *tetanus*, trismus is an early symptom, there is little or no fever, and there is often a history of an injury. If there is doubt as to the existence or nature of meningitis, lumbar puncture (p. 78) may be practised, and the fluid withdrawn should be subjected to bacteriological examination.

Prognosis.—This is very serious. Acute cases often die within a period of days or weeks, but cases that survive for some weeks may recover. In other instances, the duration may be months or even years. Death may be due to exhaustion, interference with respiration, bedsores, cystitis, etc.

Treatment.—Perfect rest of mind and body is important. The bedroom should be quiet and darkened. Leeches and hot fomentations may be applied to the spine. A hot bath or pack, a purge, and a diaphoretic are also advisable at the outset. Mercurial inunction should be carried out until the

gums are slightly touched. Anodynes may be required to relieve the pain and spasm. If the acute stage is survived, counter-irritation, friction, electricity, tonics and change of air are indicated.

Chronic Internal Meningitis.

This may be either *chronic internal pachymeningitis* or *chronic leptomeningitis*. It is most common in adult males, and among the recognised causes are repeated exposure, injury, disease of the adjacent membranes, bones or cord, alcoholism and syphilis. *Internal hæmorrhagic pachymeningitis* (*hæmatoma of the spinal dura mater*) is specially common among the insane, and is frequently associated with a similar intracranial lesion.

Morbid Anatomy.—The membranes are thickened and opaque. The nerve roots are at first swollen and afterwards atrophied. The superficial layers of the cord are often softened at first, and afterwards sclerosed, with the result that secondary degeneration takes place, especially above the lesion. The inflammation which begins in the soft membranes is often extensive. That which begins on the inner surface of the dura is often localised and associated with great new formation of tissue (*hypertrophic internal pachymeningitis*). This is most common in the cervical region (*hypertrophic cervical pachymeningitis*), and the cord as well as the nerve roots may be seriously compressed. In the *hæmorrhagic* form a great part of the inner surface of the dura is covered with a brownish exudation consisting of blood and inflammatory exudation. Syphilis may cause either pachymeningitis, or a localised leptomeningitis with gummy, caseous and fibrous material.

Symptoms.—These include pain and stiffness in the back, the former being increased by movement and pressure. An important symptom is the occurrence of severe pains radiating along the nerves whose roots are irritated. There may be hyperæsthesia, paræsthesia, and muscular twitchings. After a time, perhaps months, symptoms of damage to the nerve roots supervene—impaired sensation, muscular

weakness and wasting, and loss of reflex action. Still later, increasing pressure on the cord gives rise to weakness of the legs and perhaps to other symptoms which will vary according to the region involved.

Prognosis.—Many cases ultimately recover, though the pains may persist for a long time. The prognosis is relatively favourable in syphilitic cases, but in all varieties there is the risk that cicatricial contraction of new-formed tissue will cause permanent damage to the nerve roots, or to the spinal cord, or to both.

Treatment.—This includes rest, anodynes, counter-irritation, mercury and potassium iodide internally, electricity and tonics.

External Meningitis.

External meningitis includes two principal types, the one being primary, acute and diffuse, while the other is secondary, chronic and localised. The inflammation involves both the dura and the connective tissue between it and the bone.

The *acute* form occurs in weakly subjects, sometimes after exposure, and the pus fills the space between the dura and the bone, and may burrow among the neighbouring muscles.

The symptoms resemble those of internal meningitis (pain in the back, radiating pains, rigidity and hyperæsthesia), with serious interference with the functions of the cord, and great constitutional disturbance. The disease is almost invariably fatal.

The more common *chronic* form is generally due to extension from adjacent disease, chiefly spinal caries, and is usually confined to the neighbourhood of the primary lesion. The outer surface of the dura is covered by a layer of material consisting of pus, caseous débris, and sometimes new-formed fibrous tissue. This layer may be of considerable thickness, and is the usual cause of compression of the cord in spinal caries.

Any symptoms that may be present are scarcely to be distinguished from those of the cause (caries, etc.) on the one hand, and of compression of the cord on the other. In

cases due to caries, the prognosis practically depends on the effects produced on the cord.

Treatment has to be considered chiefly in connection with the chronic disease. It includes rest, counter-irritation, tonics such as cod-liver oil, and perhaps extension and laminectomy.

iii. Spinal Meningeal Hæmorrhage

(Hæmatorrhachis. Meningeal Apoplexy).

In *spinal hæmorrhage* the blood may be effused outside the dura mater (*extrameningeal*), or within the dura (*intrameningeal*). The latter form may be either *subdural* or *subarachnoid*.

Meningeal hæmorrhage is often due to injury. It also occurs in hæmorrhagic diseases such as purpura, and in severe convulsive disorders such as epilepsy. It is sometimes produced in new-born children by the rupture of vessels at the time of birth, but the blood in such cases has often simply descended from the cranial cavity. Occasionally the hæmorrhage is due to rupture of an aneurysm of the basilar or vertebral artery or of the aorta.

Symptoms.—Occasionally, as in convulsive diseases, the condition does not give rise to definite symptoms; but as a rule there are symptoms like those of meningitis. The attack is generally inaugurated by sudden severe pain in the back, with paroxysmal radiating pains, muscular spasm, especially in the back, and retention of urine. The symptoms are followed after a short time by considerable loss of power and sensation below the level of the lesion. The symptoms will naturally vary in different cases according to the level of the lesion. Head symptoms are absent.

Diagnosis.—In *meningitis* the onset is acute rather than sudden, and fever is present from the first. Lumbar puncture will probably yield blood in a case of hæmorrhage. In *hæmorrhage into the cord*, paralytic symptoms are likely to be prominent from the outset, whereas in meningeal hæmorrhage irritative symptoms come first.

Prognosis.—Death usually occurs in a few hours, but sometimes not for days. If the patient lives for some days

meningitis may ensue, but this too may be safely survived. Death is often due to interference with respiration, but may be due to exhaustion.

Treatment.—The patient should rest on his face or side. Icebags and leeches should be applied along the spine. Ergotin should be injected hypodermically as a hæmostatic, and morphine as an anodyne. If life appears to be threatened, it might be well to open the spinal canal, turn out the blood and apply a local hæmostatic.

iv. Myelitis.

In accordance with the opinion which has long been generally accepted, paralysis from coarse disease of the spinal cord is often of inflammatory and seldom of vascular origin, while it is certain that exactly the converse is true in the case of the brain. But though the inflammatory theory of softening of the spinal cord is still the one which prevails, it has been maintained, for instance by Bastian, that spinal and cerebral softenings should be alike regarded as due to vascular disease. Bastian further holds that thrombosis rather than embolism is the principal factor in the production of spinal softening, though he admits that in many cases of this condition no vascular occlusion can be detected. There is no doubt that cases are met with which, by their mode of onset, or by the circumstances under which they occur, irresistibly suggest a vascular origin; but in the meantime the usual nomenclature will be accepted in these pages. It should be added that there is a tendency at the present day to set aside the conception of a *chronic myelitis*, and to classify cases which were formerly supposed to be of this nature under other headings, such as insular sclerosis, syphilis, etc.

Myelitis may be classified as *acute*, *subacute* or *chronic*, according to the mode of onset. It may also be classified according to the distribution of the lesion. Thus, *transverse* myelitis involves the whole thickness of the cord for only a short vertical distance. In *focal* myelitis, there is one small focus of disease. In *disseminated* myelitis, there are many such foci scattered throughout the organ. In *diffuse*

myelitis, there is continuous involvement of a large extent of cord. When the grey matter alone suffers, the condition is called *poliomyelitis*. The expressions *central*, *peripheral*, *parenchymatous* and *interstitial* myelitis explain themselves.

Acute Myelitis—**Etiology.**—Apart from anterior poliomyelitis, which is separately described, myelitis is most common in the period from later childhood to middle age. Males suffer more than females. It may follow exposure, especially when the body is heated, or violent exertion of the muscles of the back. It may be caused by the organisms or toxins of infectious diseases, including small-pox, measles, enteric, typhus, syphilis and gonorrhœa. Injury or disease of the spine, or of the cord itself, may induce acute myelitis. Gout and alcoholism appear to be occasional causes.

Morbid Anatomy.—At the seat of inflammation the pia mater is red and the cord is swollen and softened. Softening and disintegration result also from vascular occlusion. The softening may be *red* from extravasation, *brown* or *yellow* at a later stage from change in the blood pigment, or *white* from relative defect of blood. Where there is much extravasation of blood the condition is termed *hæmorrhagic myelitis*. The microscope shows compound granular corpuscles, red blood corpuscles, leucocytes, drops of myelin, broken axis-cylinders, and dilated bloodvessels. The nerve cells become swollen and granular, and may lose their processes. As time goes on the interstitial tissue increases, and the spider cells or cells of Deiters become numerous. Secondary degeneration takes place above and below the lesion (ascending and descending degeneration).

Symptoms.—*Acute transverse myelitis* is the most common form, and is apparently due chiefly to thrombosis and softening. At the outset there may be headache, shivering and other general symptoms, and perhaps some tingling or other sensory disturbance in the limbs. These phenomena are quickly followed by weakness of the legs, which in the course of some hours, or perhaps days, may become quite powerless. As the disease is most common in the dorsal cord, the paralysis most commonly involves the lower limbs and lower part of the trunk (*paraplegia*). Sensation is lost

below the level of the lesion, in part or entirely. Voluntary control over the bladder and rectum is lost. There is incontinence if the centre in the cord is damaged, and retention (unless the transverse lesion is total) if the lesion is above the centre. But incontinence may be really an overflow incontinence. If the centre is involved, cystitis is very apt to ensue, partly owing to trophic changes and partly in consequence of catheterism. The state of the deep and superficial reflexes and of muscular nutrition depends on the level of the lesion. If the reflex centres are damaged, reflex action is lost. Muscles whose motor nerves start from cells in the inflamed piece of cord undergo wasting, and show changes in their electrical reactions. Owing to the impaired trophic state of the skin and other tissues, bed-sores readily form over the sacrum and other parts which are the seat of pressure. The temperature of the paralysed limbs is often elevated one or two degrees for a time, but is afterwards low. At the upper limit of the paralysis there may be a zone of hyperæsthesia and a sense of constriction (*girdle sensation*) owing to irritation of nerve fibres.

If the lesion is well above the lumbar enlargement, the reflexes in the lower limbs will be increased, unless the disease involves the whole thickness of the cord, in which case the deep reflexes are abolished (see p. 679). As a rule, however, the knee-jerks are increased, ankle clonus is present, and the plantar reflex is extensor.

If the patient survives, secondary degeneration ensues, and in cases where the disease is above the lumbar enlargement, phenomena develop which point to a lesion of the upper neurons of the motor path (pyramidal tract). The knee-jerks are exaggerated, ankle clonus is present, there is tonic spasm or rigidity of the muscles, the plantar reflex is extensor in type (Babinski's sign), compression of the foot causes the limb to be drawn up, and yet voluntary movement in the lower limbs, like sensation, and like control over the evacuations, may be entirely absent.

If the lesion is in the lower cervical region, the upper limbs are paralysed as well as all parts lower down ; there may be contraction of the pupils and narrowing of the

palpebral fissure ; priapism and great elevation of temperature may occur, and respiration is seriously hampered by paralysis of the intercostal muscles.

Acute diffuse or *acute general myelitis* is much less common than the variety which has been described, and is probably truly inflammatory in its nature. It may result from exposure, infectious diseases, injury or tumour. It may be accompanied by optic neuritis. The general symptoms may be severe, including high fever, a dry tongue and delirium. There is rapid loss of motion, sensation and reflex action, with paralysis of the bladder and rectum, muscular wasting and bedsores. The symptoms usually begin as paraplegia, but the paralysis spreads upwards (*ascending myelitis*), and in the most severe cases may cause death within a week.

Disseminated myelitis is often subacute in onset, and is specially common in syphilitic subjects. It may be possible to recognise by the symptoms that damage has been done to various regions of the cord. The different foci of disease may develop one after another. Constitutional symptoms are often absent in this form.

Hæmorrhagic myelitis is characterised by the sudden onset of severe symptoms in the course of what appears to be the early stage of ordinary myelitis.

Course.—Some cases recover, either quickly or slowly. Many become chronic, and such may regain much sensation and a certain amount of motion though with a spastic condition of the legs (*spastic paraplegia*). Many cases die from respiratory paralysis ; from acute respiratory diseases, which are aggravated or promoted by respiratory palsy ; from exhaustion or septic poisoning due to bedsores ; and from septic disease of the kidneys (pyelonephritis, etc.) secondary to cystitis.

Diagnosis.—In *multiple neuritis* the bowels and bladder are rarely involved, acute trophic lesions do not occur, anæsthesia is not complete, the nerves and especially the muscles are tender, and a cause is often recognisable. The four limbs may be affected, or the upper limbs without the lower. The onset is often gradual.

Ascending myelitis is distinguished from *Landry's paralysis* by the more marked involvement of sensation and of the sphincters, and by the muscular wasting, electrical changes, bedsores, and severe general symptoms.

In *spinal meningitis* there are radiating pains, pain in the back, hyperæsthesia and spasm, showing considerable irritation of nerve roots.

In *meningeal hæmorrhage* there are symptoms similar to those of meningitis, but of sudden onset.

Hæmorrhage into the cord causes sudden and severe pain, with rapid loss of power.

In *hysterical paraplegia* bedsores, incontinence, girdle sensation, marked ankle clonus, and an extensor type of plantar reflex are all absent. The plantar reflex is often absent altogether, and if present is flexor. There may be other evidences of hysteria, or the patient may have suffered from previous hysterical attacks. It must not be forgotten, however, that organic nervous disease is one of the causes of hysterical phenomena, and though the latter are clearly present, it is still necessary to make sure that no other nervous disease is present. Complete anæsthesia of the lower limbs bounded by their junction with the trunk is functional. The paraplegia of hysteria may or may not be accompanied by anæsthesia.

Treatment.—The patient should rest on the face or side, and have leeches or cups and fomentations applied over the spine. A water-bed and the most careful attention to cleanliness are essential, to minimise the tendency to bedsores. When these form they must be frequently and carefully cleansed and dressed, and the patient's posture should be changed from time to time to relieve pressure on the affected part. By way of prevention, the skin of parts on which the body rests must be regularly cleansed, dried, and dusted with zinc or starch powder. The application of spirit helps to harden the skin, but instead of hardening it, it is perhaps better to anoint it with glycerin mixed with boracic acid to the thickness of cream.

A diaphoretic and a purge should be given at first. Mercury should be rubbed into the skin at short intervals

until the gums are slightly influenced. The bladder must be attended to morning and evening, and the practitioner must never be induced by the mere fact that the urine is constantly dribbling away to neglect a regular examination of the abdomen, since the condition is not unlikely to be an overflow incontinence.

In the later stages, if life is prolonged, tonics and massage are indicated. Electricity is also indicated if the paralysed muscles are wasted and flaccid, but not if they are the seat of spasm.

Chronic Myelitis may be *transverse, focal, disseminated*, or *diffuse*. It may be the sequel of an acute attack or may be gradual from the commencement. It must be clearly distinguished from insular or disseminated sclerosis on the one hand, and from the degenerative system diseases on the other.

Etiology.—The causes include chronic alcoholism, syphilis, gout, repeated exposure, injury and neighbouring inflammation. The alcoholic form is usually accompanied by chronic meningitis.

Morbid Anatomy.—The affected white matter is grey in colour, and in old-standing cases there is shrinking. The interstitial tissue is greatly increased, the walls of the vessels are thickened, and the nerve fibres are narrowed or destroyed. Secondary degenerations are present. The pia mater over the inflamed part is thickened.

Symptoms.—The most common form is transverse or focal myelitis in the dorsal region, and this gives rise to spastic paraplegia of very gradual onset. There may be some tingling, pain, or impairment of sensation in the legs, with girdle sensation, and imperfect control over the bladder and rectum.

Muscular wasting and loss of reflex action will be present if the lumbar or cervical enlargement and the reflex centres are involved in the lesion.

The disease usually advances very slowly during a period of years, but may at any time become stationary or undergo an acute or subacute exacerbation.

Diagnosis.—In *primary lateral sclerosis*, in which spastic paraplegia is also present, there is no involvement of sensation. In *paraplegia from compression*, the cause of the com-

pression can usually be recognised, and there are frequently radiating pains owing to irritation of nerve roots. *Pachymeningitis* affects especially the cervical region, and is apt to be associated with more pain and anæsthesia than myelitis.

Treatment.—The general health must be kept in the best possible state. A change of air may be desirable. Fatigue, exposure, alcohol and all excesses must be avoided. Counter-irritation over the spine should be employed from time to time, and may take the form of blisters or the actual cautery. A daily hot douche may be employed to the back. Among the drugs recommended are arsenic, iodide of iron, and red iodide of mercury in small doses.

Acute Anterior Poliomyelitis (Acute Atrophic Spinal Paralysis. Infantile Paralysis. Essential Paralysis of Children).

Definition.—An acute disease, associated with a lesion in the anterior horn of the spinal cord; occurring chiefly in the first three years of life; often setting in with constitutional symptoms; and characterised by loss of power in a number of muscles, some of which undergo rapid wasting, whilst others recover.

Etiology.—The majority of cases occur in the first three years of life.[1] In the first ten years of life the sexes suffer equally, but the exceptional cases which occur after ten are almost all males. The disease is much more common in the hot than in the cold season of the year. It occasionally occurs in epidemic form, and may then attack more than one member of a family. An epidemic has been observed to attack the lower animals as well as human beings. The disease has been attributed to cold, injury and infectious diseases, but in a large proportion of cases the child when attacked is in apparently perfect health. The disease is itself to be regarded as an infection, and the infective agent

[1] Sir Walter Scott's right leg was paralysed by this disease when he was about eighteen months old. (See his Autobiography in Lockhart's 'Life.')

appears to act by causing thrombosis in the branches of the anterior spinal arteries, which supply the anterior horn. It has been suggested that the virus is the same as that which causes the polioencephalitis of childhood (infantile hemiplegia).

Morbid Anatomy.—The essential lesion is in the anterior grey cornu, and is characterised in the early days of the illness by thrombosis of the arteries supplying the territory involved, necrosis and softening of the tissue thus deprived of its blood-supply, complete destruction of the nerve cells, and great extravasation of leucocytes. Degenerated fibres are also found in the anterior commissure, ground fibres, root-zone, antero-lateral ascending tract, direct cerebellar tract, posterior root, and peripheral nerves. At a late stage, the anterior horn is shrunken; the interstitial tissue is increased; the motor cells are scanty or absent; and the anterior nerve roots, motor nerve fibres and corresponding muscle fibres are atrophied. The lesion therefore involves principally the neurons of the lower segment of the motor path. It destroys the cells, and accordingly the axons degenerate, while the muscular fibres to which the axons are distributed undergo atrophy.

Symptoms.—At the outset, as a rule, there are general symptoms, such as fever, headache, vomiting and anorexia. Sometimes there are severe pains in the limbs and occasionally convulsions. In some instances, however, there is neither pain nor constitutional disturbance. In a few cases the paralysis follows over-exertion, a fright, or some accident such as a fall.

The paralysis develops quickly, and is to all appearance quite random in its distribution in the limbs, except in so far as the lower limbs suffer much more frequently than the upper. It may involve all four limbs, or may be confined to a portion of one limb. As a rule, however, more than one limb suffers. The maximum palsy is quickly reached in most cases. It may be attained within a few hours or days, but is often reached before the weakness is discovered. In a few cases it is sudden, and therefore probably due to hæmorrhage. In any case, the affected muscles are quite

flabby from the beginning, and in a week or two are found to be wasted, and to have undergone electrical changes. There is usually no pain, but the skin of the paralysed part is cold and blue. Reflex action suffers if the lesion involves the corresponding reflex centre.

The cranial nerves, the sphincters, and sensation almost always escape. Bedsores do not occur. Bronchitis is an occasional dangerous complication in the early stage.

After a time, a certain amount of recovery almost always takes place, through cells which have been partially damaged, or compressed by inflammatory exudation, regaining their function; but recovery is rarely complete. The further growth of the bones of the affected limb is interfered with. The skin is cold and livid, probably in consequence of damage to vasomotor nerve fibres in the cord. The unaffected muscles undergo shortening, and in this way, aided by gravity, cause deformities. Joints which are partly supported by tendons which pass over them may be dislocated.

A fatal ending and a second attack are both very rare.

Diagnosis.—This depends on the acute onset of general symptoms, and the immobility, flabbiness, and wasting of a number of muscles in one or more limbs. The principal risk is that of mistaking the disease in its initial stage for one of the more common acute infections, and indeed, until the paralysis is developed, this is excusable. The pains might be regarded as rheumatic, but their occurrence apart from movement and apart from the joints should suggest a nervous origin. After a week, some of the paralysed muscles will be found to have lost their faradic irritability. Primary myelitis in children, as Gowers remarks, is always poliomyelitis.

In *cerebral palsy*, the deep reflexes are preserved, faradic irritability is preserved, and muscular wasting, if present, is not severe.

The various forms of *muscular dystrophy* set in gradually.

Joint disease (*e.g.*, *morbus coxæ*) should present no difficulty to a careful observer, since in this condition the pain is induced or aggravated by movement, mobility is diminished, and swelling may be present.

Prognosis.—This will be assisted by an electrical examination of the affected muscles a fortnight after the disease sets in. Early and extensive loss of faradic irritability points to serious and extensive wasting. Complete recovery cannot be expected in any case, and deformities and defective growth of the limb may result; but on the other hand, there is almost no danger to life, and great improvement may be expected.

Treatment.—At the outset the patient should be kept at rest on his side, and fomentations should be applied over the affected region of the cord. A diaphoretic and purgative draught should be administered. After the acute stage is past, fresh air, good food, tonics (including quinine, iron and strychnine), and systematic daily rubbing and passive movement of the paralysed muscles are the principal remedies. Weakened muscles should be educated by regular exercise. Galvanism should be applied to the paralysed muscles during a considerable period of time, in order to favour the preservation of their power of responding to motor impulses, if the degenerated cells and fibres should ever become capable of transmitting them. The muscles should be repeatedly stroked with the negative terminal in the form of a sponge wetted with salt solution. Deformities must be prevented, and this may require the use of special apparatus. Deformities already present when the patient comes under observation may sometimes be improved by surgical measures, such as tenotomy and tendon-grafting.

Subacute and Chronic Anterior Poliomyelitis (Subacute and Chronic Atrophic Spinal Paralysis).

There is little doubt that in the past this designation has often been wrongly applied; and especially in cases of multiple neuritis, where the lesion is not in the cord, but in the nerves. In such cases we might expect a stricter symmetry of the paralysis than is usual in poliomyelitis. Moreover, there might be tenderness of the nerves and muscles; and the history might point to a recognised cause of neuritis.

In a second group of cases, the disease ultimately proves to be exactly like progressive muscular atrophy, and deserves to be so called; but its onset, instead of being gradual, is subacute. In a case of this kind recently under my care, it was found that the anatomical as well as the clinical phenomena were intermediate between those of anterior poliomyelitis and progressive muscular atrophy.[1] The distinguishing feature of this group is the progressive tendency.

A third group resembles genuine acute anterior poliomyelitis in every respect except that its onset is subacute rather than acute, and that it is more common in adults than in children. There is constitutional disturbance at the commencement, and the paralysis, after being relatively widespread, recedes to some extent, though not to the length of complete recovery.

v. Acute Ascending Paralysis

(Landry's Paralysis).[2]

Definition.—A disease characterised by motor paralysis which spreads from the lower limbs to the trunk, upper limbs and respiratory muscles; attended by a high rate of mortality; and not referable to any known anatomical changes.

Etiology.—The disease is most common in the first half of adult life. Males suffer more than females. Alcoholism, syphilis, acute infections, wounds, and exposure are among the causes.

Pathology.—No constant lesion has been discovered in the brain, spinal cord, or peripheral nerves. The spleen is occasionally enlarged. Many hold that the disease is an acute multiple neuritis, a view which is quite in accordance with the little that is known of the etiology. Changes have been found in the anterior horn cells and peripheral nerves, and in the meantime the disease may be looked upon as an acute poisoning of the lower motor neurons.

[1] Monro and Findlay, *Trans. Glasg. Path. and Clin. Soc.*, December, 1905.

[2] Described by Landry in 1859.

Symptoms.—In some instances there are premonitory symptoms such as feverishness, malaise and numbness in the extremities. Of the characteristic symptoms, the first is weakness of the legs which rapidly progresses, so that in a few days or even hours, their power is completely lost. The palsy extends up to the abdomen, chest and upper limbs and often to the neck, so that the patient cannot move about in bed, defæcation is rendered difficult, and respiration, deglutition and articulation are embarrassed. The affected muscles are flaccid but do not waste, and the electrical reactions of nerves and muscles are normal. Reflex action is lost along with muscular power. Sensation is little if at all affected, and there is no pain. There are no bed-sores. The sphincters escape. The cerebral functions are seldom disturbed. The spleen may be enlarged, but there is seldom pyrexia.

Death usually results from asphyxia, but is sometimes due to cardiac paralysis.

Prognosis.—The majority of cases end fatally, many of them in less than a week. Some cases advance more slowly and last for several weeks. In a minority of cases, estimated at about a third, the disease is arrested after it has involved the arms, occasionally at an earlier stage, rarely when the medulla is affected. After remaining stationary for a time, the paralysis gradually recedes in the reverse order to that in which it originally spread, but many weeks may elapse before the power of the lower limbs is completely restored.

Treatment.—This is similar to that of myelitis. Any cause should be neutralised if possible, *e.g.*, by salicylates in cases due to cold, by mercury and iodide in syphilis, and by perchloride of iron or some antitoxic serum in septic cases. Recovery has followed the use of ergot (Gowers).

vi. Caisson Disease

(Diver's Paralysis. Compressed Air Illness).

Persons who, like divers or workers in caissons, are subjected to much more than the normal atmospheric pressure, sometimes suffer from troublesome symptoms immediately

or at a short interval after their return to the normal atmospheric pressure. The condition is due to the frothing off from the blood, during decompression, of air which has accumulated in it under the abnormal pressure. The air-bubbles thus produced obstruct the small bloodvessels, and in this way, as well as by escaping into the tissues by rupture of the vessels, account for the symptoms.[1]

Morbid Anatomy.—The usual change found after death is congestion of the brain, spinal cord and other organs. Occasionally air has been seen in the veins. If death is postponed for several weeks, the spinal cord may be softened, especially in the lower dorsal or lumbar region, and pyelitis, cystitis and bedsores may be present.

Symptoms.—Pains in the limbs, and especially about the knees, are very common, and occur much more readily than paralysis. These pains are known by workmen as 'bends.' They vary in severity and in duration, and are sometimes of agonising intensity.

The usual form of paralysis is paraplegia, with involvement of motion, sensation and the sphincters. The palsy may be slight and pass off in hours or days; or it may be rapidly fatal, or, again, it may continue for months. Occasionally there is transient hemiplegia, and in some instances coma with death in a few hours.

Prognosis.—The great majority of cases recover.

Treatment.—To prevent this illness, the pressure in the lock through which the workmen return to the outer air should be reduced very gradually; thus, one minute for each 5 pounds of pressure has been found satisfactory. When symptoms set in, recompression is indicated; and for this purpose a medical lock fitted with beds should be attached to the works. Morphine may be required to relieve the pains.

[1] For every 10 metres or 33 feet of depth below the surface of the water, 1 (additional) atmosphere, or + 15 pounds of air pressure, is required to keep the water out of the caisson or diving bell. At a depth of 100 feet, therefore, the pressure is 3 atmospheres. This is about the maximum reached in recent times, but the experiments of Leonard Hill appear to show that men may work in safety at depths up to 200 feet, provided that the subsequent decompression is carried out sufficiently slowly.

vii. Hæmorrhage into the Spinal Cord (Hæmatomyelia).

Etiology.—This condition is excessively rare when compared with hæmorrhage into the brain, perhaps because the long tortuous course of the vessels saves them from the high pressure which favours degeneration. It is most common in the first half of adult life, and males suffer much oftener than females. Injury is the most common immediate cause, but hæmorrhage may occur in connection with myelitis, tumours of the cord, syringomyelia, tetanus and other convulsive disorders, or without obvious reason. It usually begins in, and is often confined to, the grey matter. The lower cervical region is the part most frequently involved.

Symptoms.—The symptoms set in suddenly with severe pain in the back, loss of power and feeling below the level of the lesion, and loss of control over the sphincters. As the one side of the cord is commonly affected more than the other, there is frequently motor loss on one side, with sensory loss (particularly with regard to pain and temperature) on the other. Occasionally the symptoms take an hour or two to reach their height. Thereafter they resemble those of an acute focal myelitis.

Diagnosis.—In *meningeal hæmorrhage*, the symptoms point, for a time at least, to irritation rather than to destruction of nerve fibres. If cord symptoms have preceded the sudden onset, *myelitic hæmorrhage* is probable. In *acute anterior poliomyclitis*, pain is often absent, sensation is preserved, and constitutional symptoms may be present at the commencement.

Prognosis.—This is very grave until the symptoms begin to decline. Thereafter it must be estimated as in myelitis. As a rule, recovery is at best incomplete.

Treatment.—In the early stage, absolute rest in the prone position, the application of leeches and icebags to the spine, and the hypodermic injection of ergotin and, in some cases, of morphine are indicated. The subsequent treatment is that of myelitis.

viii. Compression of the Spinal Cord.

Etiology.—The most common cause is caries of the vertebræ with accumulation of pus and caseous débris between the bone and the dura mater; with this mechanism, displacement of the bone may co-operate. Growths in the spine or in the membranes, aneurysms which have eroded the vertebræ, and the thickened dura mater in pachymeningitis are other compressing agents. Compression causes not only narrowing of the cord, but also local myelitis with secondary ascending and descending degeneration.

Symptoms.—Three kinds of symptoms may be recognised: (1) symptoms pointing to disturbance of nerve roots; (2) symptoms pointing to disturbance of the cord itself; and (3) in many cases, phenomena due directly to the cause of the compression.

(1) The most constant root symptom is severe pain radiating along the nerves whose roots are irritated or compressed. This is often accompanied at first by cutaneous hyperæsthesia, but later on there may be anæsthesia in combination with the pain (*anæsthesia dolorosa*). The pains are far more severe in connection with tumours of the spinal column than in caries, and they are greatly aggravated by movement. Damage to motor roots causes weakness and wasting of the corresponding muscles.

(2) The cord symptoms usually appear later. The most common is *paraplegia* of gradual onset, with increase of the cutaneous and deep reflexes. Less constantly sensation is disordered or lost, and the sphincters are affected. The other symptoms will naturally vary according to the level of the lesion, and according to the acuteness or chronicity of the secondary myelitis. The terrible root pains in malignant disease of the spine associated with the cord symptoms justify the designation *paraplegia dolorosa*.

(3) In many cases there is deformity of the spine (usually angular or Pott's curvature), and there may also be local tenderness.

Diagnosis.—This depends on the combination of root symptoms and cord symptoms, and is often corroborated by

the discovery of spinal curvature or tenderness, or of some other morbid condition—*e.g.*, a tumour—which will account for the pressure.

Prognosis.—A large proportion of cases due to caries ultimately recover completely. In malignant disease the outlook is of course hopeless, and in aneurysm it is scarcely any better.

Treatment.—Rest in the recumbent position is often sufficient for cure, especially in children, when combined with the administration of cod-liver oil, and perhaps the iodide of iron. If the symptoms do not promptly begin to subside, continuous extension should be employed; and if after months no improvement occurs, and still more if the case is becoming worse, laminectomy should be performed. The usual constitutional treatment should be employed in tubercular cases. In malignant cases anodynes will probably be required in addition to the ordinary treatment of myelitis.

ix. Tumours of the Spinal Cord and its Membranes.

Etiology.—Cancers and sarcomata of the vertebræ may invade the extra-dural space, and in rare cases a growth (generally lipomatous or parasitic) originates in the extra-dural tissue. These two *extra-dural* groups, however, will not be further described here.

Of tumours which commence in the *membranes*, within the sheath of the dura mater, the most common are the syphiloma, the sarcoma and the myxoma. Neuromata and sarcomata may grow on the *nerve roots*, and are often multiple. Tumours arising in the substance of the *cord* are not so common as meningeal growths. The 'solitary tubercle' is the most frequent tumour of the cord, and among others are the syphiloma, glioma, sarcoma, and myxoma.

The tumour generally grows from the pia mater or from the tissue around the central canal. Tubercular growths usually originate in the grey matter.

Morbid Anatomy.—*Tumours outside the cord* compress the nerves, compress the cord, and excite myelitis which leads to secondary degenerations.

Tumours in the cord cause local swelling of the cord; myelitis with secondary degenerations; sometimes hæmorrhage; and sometimes obliteration of the central canal at the seat of disease with dilatation (*hydromyelia*) higher up.

Symptoms.—A *tumour outside the cord* gives rise to root symptoms, and after a time to cord symptoms, as described in connection with compression. The root symptoms are often at first unilateral, and include radiating pains, with hyperæsthesia or anæsthesia (*anæsthesia dolorosa*), girdle sensation, muscular spasm and muscular atrophy. The cord symptoms include paralysis with features which vary according to the level of the lesion. The palsy usually sets in gradually and spreads gradually, but is sometimes more rapid in development owing to the occurrence of myelitis.

A *tumour in the cord* causes gradual loss of motion and sensation below the level of the lesion. If it begins in one side of the cord, motion may at first be lost on the same side and sensation on the opposite side. Ultimately the growth tends to involve the whole thickness of the cord, either by actual invasion or by the agency of myelitis. The root symptoms are usually less marked, and the cord symptoms earlier in appearing, than in tumours of the membranes.

In both classes of tumours, pain is usually the first symptom. It radiates along the nerves derived from the cord at and below the level of the lesion, and is often of frightful intensity. As already stated, it is often unilateral for a time. Loss of sensation develops in the same way as pain. If the tumour begins in the membranes, there may be pain and tenderness in the spine. There may be a marked girdle sensation, with hyperæsthesia of the skin at a level corresponding to the growth. The pain may be increased by movement, but not to the same extent as in tumours of the vertebræ.

Muscular spasm is specially common when the tumour grows from the membranes. It may involve the muscles of the back at the level of the lesion, the abdominal muscles at the level of the girdle sensation, and the muscles of the lower limbs. As in the case of pain, the symptom may be manifested in parts innervated from the cord below the level

of the disease, so that it may be due to irritation of tracts in the cord as well as to irritation of nerve roots. Paralysis is of gradual onset, often unilateral at first and slow in progress, though frequently complicated by subacute or acute myelitis. Muscular wasting results either from damage to the anterior nerve roots, or from the disease involving the anterior cornu.

If the disease is entirely above the lumbar enlargement, there is marked increase of both the cutaneous and the deep reflexes.

Control over the sphincters is often lost along with the power in the lower limbs. Bedsores are common in the late stages of the disease.

A tumour in the *cervical region* causes symptoms referable to the neck and the upper limbs ; sometimes changes in the pupil ; weakness, and exaggeration of reflexes in the legs ; and, if unilateral, weakness of the limbs on the side of the lesion with anæsthesia on the opposite side. In tumour of the *dorsal region*, the root pains and the loss of the cutaneous reflexes indicate the level of the lesion ; and if neither tumour nor inflammation has invaded the lumbar enlargement, the deep reflexes in the legs are increased. In tumour of the *lumbar enlargement*, the root pains and muscular atrophy involve the legs, and the reflexes are lost. The symptoms in tumour of the *cauda equina* are generally bilateral from the outset.

Diagnosis.—This depends on the presence of symptoms of a focal lesion of the cord of a slowly progressive character The evidence is much more conclusive if the root symptoms and cord symptoms of compression are present. The presence of syphilis or tuberculosis as a possible cause is of some value.

In *caries of the spine*, root pains are seldom severe, weakness of the legs is bilateral, deformity of the spine is often recognisable, and the patient may present other evidence of tuberculosis.

A *tumour commencing in the bones* is associated with pain which is greatly aggravated by movement. The growth may appear at the surface.

It is important not to regard the pains as neuralgic.

Their situation and obstinate persistence, and in the case of sciatic pains their bilateral distribution, render *neuralgia* an unsatisfactory explanation.

Sciatica is almost never bilateral.

Prognosis.—Growths outside the cord can sometimes be removed by operation. Syphilitic growths are amenable to treatment. A tubercular growth may possibly become stationary. Other tumours tend slowly to progress, though temporary arrest may take place. If myelitis has occurred, this may subside, so that actual improvement may take place for a time. But in the main the tendency is towards death, often after great suffering.

Treatment.—In syphilitic cases, potassium iodide must be given internally, and mercurial inunction should also be employed. In tubercular cases, cod-liver oil and iodide of iron are indicated. When paraplegia sets in, complete rest and careful nursing are desirable. A few cases of tumour within the spinal canal can be successfully treated by the surgeon.

x. Syringomyelia.

Cavities met with in the spinal cord may be divided into two distinct classes, both of which are included under the designation syringomyelia. (1) In the first class there are (*a*) cases of dilatation of the central canal, which is distended with fluid. This condition may be congenital, and is occasionally present in spina bifida. Cavities of this kind may have some embryonic tissue around them. Distension and dilatation of the central canal may also result from obstruction of that canal by a tumour low down. Cavities originating in the central canal are characterised by the presence of that variety of epithelium which lines the normal canal, and they constitute *hydromyelia* (*hydromyelus*, or *hydrorrhachis interna*). (*b*) In other cases the cavity seems to have originated apart from the canal, and thereafter to have established a communication with it, *e.g.*, in connection with inflammation around the canal, or by the softening of inflamed tissue. In cases where the cavities communicate with the central canal, the tissue surrounding the cavities is

very apt to resemble the embryonic tissue from which the cord was derived. In some instances, too, there is sclerosis with defect of the nerve elements around the posterior horns and in the posterior columns. As this abnormality is specially marked at the central ends of the postero-internal columns, which are among the last portions of the cord to reach complete structural development, there is little doubt that it is due to congenital defect. (*c*) It has been suggested that cavities in the cord may result from minute hæmorrhages taking place at birth in connection with difficult or protracted labour.

(2) In the second class the spinal cord is the seat of a new growth in which cavities are present. The growth is gliomatous or sarcomatous in its nature, and in some cases gives rise to an enormous increase in the size of the cord. It may extend through the whole length of the cord. The growth is probably due to multiplication of embryonic neuroglia, and is spoken of as *gliomatosis ;* while the word *gliosis* is applied to a similar condition when it does not amount to a distinct tumour. A glioma of the pons is sometimes found to be connected by tracts of gliosis with similar tissue around a congenital cavity in the cervical spinal cord. Why cavities develop in gliomatosis is not known, but they are supposed to be due to breaking down of tissue, and this in its turn is supposed to be related (and this applies also to a similar growth in the brain) to the excessive pressure under which the growths must develop within the closed cavities of the spinal canal and cranium.

Morbid Anatomy.—Cavities of considerable size may exist in the spinal cord without giving rise to symptoms. (1) Those belonging to the first class which are connected with the central canal, whether primarily or secondarily, tend to show embryonic neuroglia (gliosis) in their walls, as well as a lining of the characteristic epithelium of the central canal. As they grow in size, these cavities tend to exert pressure upon the neighbouring grey and white matter, thus giving rise to the characteristic symptoms of syringomyelia. They show a special tendency to grow into one or both of the posterior horns, but they may also damage the anterior

horns and pyramidal tracts. The cord looks unduly large to the naked eye, and may yield fluctuation to the fingers. In the process of removal from the body, the fluid will probably escape, whereupon the cord collapses and becomes ribbon-like. The Sylvian aqueduct and even the ventricles may be dilated. (2) In gliomatosis the cavities extend vertically in the cord, so as to form tubes. These sometimes divide into two or more, with the result that, as seen in transverse sections at different levels, the cavities may differ in number as well as in shape. The cavities have no epithelial lining. Both the gliomatosis and the cavities invade any part of the cord, and the symptoms they give rise to are of much more rapid evolution than those met with in the first class of cases. The cord is enormously enlarged, especially in the cervical region, and usually retains its form after removal. The growth may be found to extend upwards into the medulla.

Symptoms.—These are accounted for by the gliomatous infiltration, by secondary degeneration of the new-formed tissue, by the pressure of the fluid which distends the cavities, etc. In some cases no symptoms are complained of. In others the earliest phenomena are suggestive of myelitis, and are due to hæmorrhage into a cavity from an ill-supported or eroded bloodvessel. The more characteristic features of the disease vary considerably, and begin, as a rule, gradually in early adult life. The two most typical are dissociated anæsthesia and muscular atrophy. The first is characterised by loss of the senses of pain and temperature, with preservation of the sense of touch. These conditions are chiefly observed in the upper limbs and upper part of the trunk ; occasionally over the face and back of the head ; almost never as far down as the pubes. This curious symptom is explained by the different routes taken in the cord by the different kinds of sensory fibres ; those connected with pain and temperature pass near the walls of the enlarging cavity in the central grey matter of the cord and thus suffer from the pressure, while those connected with touch are in the white matter. The muscular weakness and wasting usually begin in the small muscles of the hands,

giving rise to the 'claw-hand,' as in chronic spinal muscular atrophy. The muscles of the shoulder and even those innervated by the cranial nerves may suffer. Lateral curvature of the spine is common. These phenomena depend upon damage to the anterior horns of the cord and to the corresponding grey matter within the cranium. Exaggeration of the knee-jerks, ankle clonus, and Babinski's sign may result from pressure of a distended central canal on the pyramidal tracts. The muscular changes are sometimes best seen in one limb, and the sensory disturbances in the opposite limb.

Among the other symptoms are trophic changes in the skin, bones and joints, and painless whitlows. Excessive redness of the skin and sweating are common. They are specially apt to involve the limb whose muscles suffer most. They may be widely distributed in cases of extensive gliomatosis with cavities. Joint lesions are rare, but are similar in their nature to the arthropathy of tabes (Charcot's joint disease). Secondary degeneration may take place and give rise to spastic paraplegia or ataxy. Owing to the analgesia, patients are apt to burn their fingers, *e.g.*, in smoking cigarettes. The sphincters generally escape. The occasional presence of nystagmus is, like paresis of the ocular or facial muscles, to be referred to changes in the region of the Sylvian aqueduct. Optic neuritis has been met with in rare cases.

Diagnosis.—This depends on the two symptoms indicated, viz., the dissociated anæsthesia and the muscular atrophy. The principal diseases from which the distinction has to be made are chronic spinal muscular atrophy (including amyotrophic lateral sclerosis) and cervical pachymeningitis.

Prognosis.—The disease is incurable, but it is often very chronic, and life may be preserved for many years without any change taking place in the clinical phenomena. The disease, however, involves increased risk from disorders of the respiratory system. Gliomatosis is much more serious than the other variety, and may cause death within a very few years.

Treatment.—The general health should be maintained at the highest possible level. The muscular atrophy should be

treated by strychnine, as in the case of chronic spinal muscular atrophy.

Morvan's Disease (*panaritium analgicum ; painless whitlows*) is not definitely separable from syringomyelia. It is sometimes regarded as a variety of that disease, in which trophic changes, and in particular painless whitlows, are specially prominent. But the few autopsies that have been made indicate the existence of neuritis in addition to the changes in the spinal cord.

The affection involves the upper limbs. Muscular weakness and wasting, neuralgic pains, loss of all forms of sensation, painless whitlows, and recurring ulcers are the principal features. The disease is extremely chronic.

xi. System Diseases of the Spinal Cord (Neuron Diseases).

(1) Locomotor Ataxy (Tabes Dorsalis. Posterior Sclerosis).

Definition.—A disease associated with primary degeneration of afferent neurons of the posterior nerve roots, and characterised, when well marked, by inco-ordination of movements, loss of muscular tone, loss of deep reflexes, pains, defective sensation, and visceral disturbance.

Etiology.—This is the most common chronic disease of the spinal cord. It is about ten times as frequent in men as in women, and usually begins between twenty and fifty. A large majority of the patients have had syphilis several or many years before, but the lesion is in no way specific. It is a parasyphilitic affection (p. 150), exactly similar to that which occurs in the small proportion of cases where syphilis can apparently be excluded. Injury and exposure to heats and colds (as in bakers) are also causes. Tabes is occasionally met with in childhood or adolescence (*infantile* or *juvenile tabes*), in which case the preceding syphilis is almost always inherited. It is now and then seen in husband and wife (*marital tabes*), the history of the double affliction starting with syphilis in the husband.

Morbid Anatomy.—There is sclerosis or grey degeneration in the posterior columns of the cord. The change is most

marked as a rule in the lumbar region, and usually tends when traced upwards to gradually confine itself to the columns of Goll. In slight cases, only the posterior root-zone may be degenerated in the lumbar region. The sclerosis is characterised by wasting of nerve fibres and overgrowth of interstitial tissue. The loss of the medullary sheaths of the nerve fibres which have wasted or vanished, and the increase of interstitial tissue, account both for the translucent grey appearance (grey degeneration), and the increased firmness (sclerosis) of the degenerated white columns. Lissauer's tract usually, and the direct cerebellar and ascending antero-lateral tracts in some cases are also affected. The posterior nerve roots are atrophied as far as the ganglia. In some cases, the afferent fibres of the peripheral nerves degenerate, chiefly at their peripheral ends. Occasionally the cells of Clarke's column and of the anterior horn undergo atrophy. The optic nerves and the nuclei of the motor cranial nerves may also suffer. Disease has been found in the posterior root ganglia which contain the trophic cells of the principal fibre systems which degenerate, but not so constantly as might have been anticipated. It is intelligible that the distal portions of the axons should show signs of degeneration sooner than the parts nearer the trophic cells or the cells themselves.

The disease is to be regarded as due usually to a syphilitic toxin which causes a premature decay of certain afferent neurons, and especially of afferent neurons from the muscles ; namely, the afferent muscle nerves which run in the peripheral nerves, and the continuations of the same in the posterior columns of the cord. The nutrition of these fibres is controlled by cells in the ganglia of the posterior roots. Frequently, however, within the cranium, and occasionally in the spinal cord, motor structures also suffer.

Symptoms.—It is possible to recognise three stages : the pre-ataxic, the ataxic, and the paralytic ; but fortunately a very large proportion of cases do not pass beyond the second stage, or the transition between the first and second stages.

One of the earliest symptoms is pain in both legs, sometimes slight and suggestive of rheumatism, but often momentary, recurring, and of great severity (*lightning pains*). The

pains may involve a portion of the limbs, or may shoot along their whole length. They may be influenced by the temperature of the atmosphere, and are very irregular in their recurrence. These brief pains may be either superficial or deep-seated. The former give rise to tenderness of the skin, even though there is analgesia at that part. The latter last slightly longer than the momentary lightning pains. Both are most common in the legs. Other pains of tabes are more persistent, and may last for hours, days, or even months. They are most common in the trunk, and are usually deep-seated, though one of them—the girdle sensation—is often superficial. They include an agonising muscular pain, and widely diffused pains or paræsthesiæ. Gowers has described a *tabid* or *tabetic neuralgia*, where the pains are the only prominent symptom, even the knee-jerk being preserved.

Sensation may be disturbed in the feet, so that the patient feels as if he were standing on a soft carpet instead of a wooden floor. There may be blunting of sensibility in the legs, especially for pain and temperature. Common muscular sensibility may be impaired or lost, so that compression or electrical stimulation of the muscles may not be perceived. Loss of the sense of posture underlies the disorder of movement known as ataxy. The conduction of sensory impressions from the legs may be delayed for a second or two. Thus, in one case a pin-prick was not felt till two seconds had elapsed, but it continued to be felt for eight seconds after the pin was withdrawn. The prick of a pin in one place may be referred by the patient to another part (*allochiria*).

The cutaneous reflexes suffer along with cutaneous sensibility.

A *girdle sensation* is common. The sense of constriction is usually about the level of the epigastrium, but may be lower or higher.

The knee-jerks are almost always lost when the patient comes under observation. The loss is attributable to damage to the afferent muscle nerves.

Inco-ordination or *ataxy* in standing and walking is a characteristic symptom in well-marked cases, but in the early

stages it is recognised only by special tests. It also is due mainly to degeneration of the afferent muscle nerves, though possibly in part to loss or retardation of afferent impulses from joints, tendons and skin. The unsteadiness is brought out, or if present is more clearly demonstrated, if the patient stands with his feet close together and shuts his eyes (*Romberg's symptom*), or if he tries to walk along the floor on a single plank, or tries to walk backwards, or turns round quickly. He may himself have noticed a similar difficulty when washing his face. In walking, if the ataxy is severe, the feet are not placed in position in an orderly manner, but are flung forwards with unnecessary force. The patient has to watch carefully where he is stepping; or, to avoid this inconvenience, he guides himself by the aid of two sticks. The excessive effort involved in this embarrassed mode of progression naturally reduces the distance to which the patient is able to walk; but if his legs are tested as he lies in bed, they will be found to possess their full power.

Another very important symptom is *hypotonus*, viz., diminution or loss of muscular tone. The normal tone or tonic contraction of the muscles, which accounts for the perceptible involuntary resistance to passive movement, depends upon the integrity of a reflex arc whose centre is in the cord. In tabes the afferent part of this arc is damaged, and the affected limbs are unduly flaccid. Like the loss of the knee-jerks, the muscular anæsthesia, and the ataxy, hypotonus depends upon damage to the afferent muscle nerves.

The bowels and bladder are usually affected. Difficulty and delay in evacuation of the bladder, retention of urine, and incontinence of urine are all common. Constipation is the rule, and may be very troublesome. The sexual functions are usually impaired.

Ocular symptoms are also common. The *Argyll-Robertson pupil* (the pupil contracting when a near object is looked at, but not when exposed to light) is almost always present (*reflex iridoplegia*). The pupils are sometimes small (*spinal myosis*), and may fail to dilate on painful stimulation of the skin of the neck (*cutaneous iridoplegia*). *Diplopia* and *ptosis* may be present, either as transient or as lasting symptoms.

Primary *optic atrophy* with restriction of the visual fields is present in a small proportion of cases, and is often early.

Visceral crises are common in connection with the stomach, and are characterised by epigastric pain with or without vomiting; the pain may amount to agony. Slight disturbances of the kind may be among the earliest symptoms. Crises occasionally involve the larynx (the features being weakness of the abductors, spasm and cough), rectum and other parts.

Trophic disturbances are seen chiefly, but not only, when the disease is advanced. *Perforating ulcer* of the foot is a chronic painless process which develops in the sole, especially over the first metatarso-phalangeal joint, often after the paring down of some callosity. The bones may become unduly brittle, so that *spontaneous fractures* may occur. *Charcot's joint disease* (*tabid arthropathy*) is characterised by enlargement of the ends of the bones, damage to the articular surfaces, and sometimes effusion, changes which in some ways closely resemble those of osteo-arthritis.[1] The condition is most common in the knee, elbow, ankle and tarsus, and is attended by remarkably little suffering. This arthropathy, occurring in the tarsus, constitutes the *tabid foot* (*tabetic foot*). It differs from most diseases of joints in respect that the mobility may be much increased. In one patient it involved the hip, knee and foot in one limb. The condition may undergo great improvement and perhaps complete cure. Loss of hair, nails and teeth, local sweating, ecchymoses and herpes may occur, sometimes in association with attacks of pain. In advanced cases, muscular atrophy may occur from invasion of the anterior horns. When this involves certain muscles which act upon the foot, a variety of club-foot is produced, known as *tabid club-foot;* this must be distinguished from the tabid foot already mentioned.

In some cases, though generally not for many years, the patient becomes unable to walk. This is due chiefly to extreme ataxy, and may be styled the paralytic stage. Occasionally an acute or subacute exacerbation of the disease develops, but this may be recovered from. General paralysis

[1] Arthropathy is occasionally observed also in syringomyelia.

of the insane and tabes dorsalis often coexist, the two having a similar relationship to syphilis. If the disease extends up to the posterior roots connected with the cervical enlargement of the cord, corresponding symptoms will arise in connection with the upper limbs. The territory of the ulnar nerve is usually first invaded, since it arises from the lower roots of those which enter the brachial plexus.

Diagnosis.—The pains in the legs are apt to be mistaken for rheumatism, but such a mistake on the part of a medical man is inexcusable. The knee-jerks and pupils must be examined, the power of standing steadily tested, and the condition of the sphincters ascertained.

Sciatica is unilateral, whereas the pains of tabes are bilateral.

In most cases of *multiple neuritis*, there is as much weakness as inco-ordination; whereas in tabes there is inco-ordination or ataxy without paralysis of the legs. In neuritis, there is often tenderness of the muscles and nerves. In exceptional cases, alcohol, arsenic and the diphtheria toxin, though usually involving motor nerves, give rise to ataxy, with loss of the knee-jerks, and without motor palsy. In these rare cases the muscles may possibly be tender, and the reflex iridoplegia and involvement of the bowels and bladder are likely to be absent. If the history can be obtained, it may explain the case at once.

In *ataxic paraplegia*, the knee-jerks are preserved.

Hereditary ataxy begins in early life, and is accompanied by nystagmus, changes in articulation, spinal curvature and club-foot.

In *cerebellar disease*, the inco-ordination makes the patient reel like a drunken man ; the ataxy is quite different from that of tabes. Moreover, the knee-jerk is usually present, and there are often head symptoms such as headache, vomiting and optic neuritis.

The risk of misinterpreting the pains in the legs has been alluded to ; the same warning is applicable with regard to attacks of gastric disturbance in men.

Prognosis.—Complete recovery is not to be expected, but arrest of the progress of the disease in the early or pre-ataxic

stage is extremely common, and even patients who are seriously disabled and suffer terribly may improve greatly. A patient may recover so far as to get rid of every symptom that troubled him, or of every symptom except one—say a girdle sensation. Such individuals may relapse after months or years. Much will depend on their subsequent mode of life. The first stage therefore may last for many years. When optic atrophy is present at an early period, the spinal symptoms seldom advance far.

Tabes itself seldom causes death, but unless the bladder is carefully watched, and, if need be, regularly emptied by the catheter, the patient runs the risk of acquiring fatal kidney disease in consequence of retention of urine. Overflow incontinence must not be mistaken for simple incontinence. In rare cases, valvular heart disease (possibly syphilitic), trophic lesions, or laryngeal palsy may be fatal.

Treatment.—Mental and bodily fatigue and all excesses must be avoided, but the patient should continue at his usual occupation if this is practicable. Antisyphilitic remedies need not be tried unless the infection took place within a very few years. Among the drugs that may be employed with a view to arrest the progress of the disease are arsenic, strychnine, silver nitrate and belladonna. The arsenic and strychnine may be given in doses of 5 minims of the official solutions. The silver nitrate must not be used for more than a few weeks at a time in case argyria should set in.

The pains and crises may, if very severe, require a hypodermic injection of morphine to make them tolerable, but it is preferable to use acetanilide or antifebrin (a single dose of 10 grains), phenazone or antipyrin (20 grains), or phenacetin (10 grains). Superficial pains may be relieved by local applications of aconite liniment, or of chloroform on lint covered with gutta-percha tissue ; or cocaine may be injected hypodermically at the seat of pain. Gowers recommends the application for ten minutes or longer of the positive electrode of a galvanic battery saturated with cocaine solution (6 to 10 per cent.). Gowers further recommends aluminium chloride (5 to 10 grains thrice daily) as an agent which tends to diminish the frequency as well as the severity

of the pains. Ammonium chloride and the salicylates have occasionally been found helpful in this way.

Nitrite of amyl may be used for laryngeal crises. Constipation may be treated by cascara or by aloes and iron with belladonna and nux vomica in pill. The bladder must have careful attention. Once the disease becomes stationary, the patient, if he suffers from ataxy, should have a course of exercises designed to re-educate the co-ordination of movement in the limbs. Orthopædic apparatus will be required in some cases.

(2) Chronic Spinal Muscular Atrophy. (Progressive Muscular Atrophy. Wasting Palsy. Creeping Paralysis. Duchenne-Aran Type of Muscular Atrophy).

Definition.—A disease characterised by progressive weakness and wasting of muscles in consequence of degeneration of the neurons in the lower segment of the motor path.

The designation *progressive muscular atrophy* is usually reserved for cases in which the muscles of the limbs suffer first. But one form of *bulbar paralysis* is an exactly analogous condition, the affected neurons being connected with the medulla oblongata instead of with the spinal cord. Similarly *chronic progressive ophthalmoplegia* is due to degeneration of corresponding neurons connected with the pons Varolii and crus cerebri.

Charcot taught that in addition to this disease, in which the lower motor neurons are degenerated, there is another in which both upper and lower neurons are degenerated, and he gave to the latter form the name *amyotrophic lateral sclerosis*. There is good reason to believe, however, that in almost all cases both neurons are degenerated, though the degeneration of the lower neurons prevents the symptoms of degeneration of the upper neurons from becoming fully manifested. As a rule, therefore, cases that are recognised clinically as *progressive muscular atrophy* are really anatomically what Charcot would have called *amyotrophic lateral sclerosis*. The symptoms vary in different cases according

as the lesion preponderates in the upper or in the lower neurons.

Etiology.—Males suffer more than females. The disease seldom begins before the age of twenty-five. Habitual exposure, anxiety and lead poisoning are occasional causes. But in many instances no cause can be traced.

There is a rare variety of degeneration of the lower or spinal motor neurons which *occurs in families* or in isolated cases. It begins about the end of the first year, and leads to almost complete paralysis and death by the age of five or six. It forms a connecting link with idiopathic muscular atrophy, inasmuch as the wasting begins at the hip, trunk and thigh, and progresses towards the distal ends of the limbs. It may even be met with in later childhood and adolescence, and in some instances the cerebral or upper motor neurons are also involved.

Morbid Anatomy.—There is great wasting or complete disappearance of the large motor cells in the anterior horns of the spinal cord at the level corresponding to the wasted muscles. The anterior root-fibres and the motor fibres of the peripheral nerves are wasted. The muscular fibres are narrowed, degenerated or lost altogether. In addition to the changes in the anterior horns there is generally grey degeneration of the crossed and direct pyramidal tracts (the lesion of both neurons corresponding to *amyotrophic lateral sclerosis*). This can often be traced up as high as the bulb, and has sometimes been traced to the origin of the upper neurons in the cortex. There is frequently atrophy of the cells in the motor nuclei of the medulla (*bulbar paralysis*), corresponding to the atrophy of the cells in the anterior cornua of the cord.

Symptoms.—The disease almost always shows itself first in the upper limbs, usually in the hands, but sometimes in the shoulder muscles. There is very gradual wasting of the muscles with weakness in proportion to the wasting. The intrinsic muscles of the hand suffer early, so that the thenar and hypothenar eminences and the interosseous tissues waste, and the bones become conspicuous. Adduction and abduction of the digits and opposition of the thumb are

impaired or lost. Under the continuous tonic action of the long muscles, which are no longer resisted by the small muscles, over-extension is induced at the metacarpo-phalangeal joints with flexion at the interphalangeal articulations, so that the *claw-hand* (*main en griffe*, *griffin-claw*) is evolved. The thumb tends to be drawn back into the same plane as the other digits, so that the hand resembles that of an ape. The long muscles of the digits and of the wrist suffer to a less extent. The muscles of the shoulder girdle and upper arm often suffer early, beginning with the deltoid.

The affected muscles show an increase of mechanical irritability, so that when they are struck, a bundle of fibres contracts. They are often the seat of *fibrillation*, a spontaneous twitching of small bundles of fibres. Fibrillation may precede wasting. Galvanic and faradic irritability gradually declines as the muscular tissue wastes. The tendon-reflexes in connection with the atrophied muscles disappear.

The disease spreads to the muscles of the neck, and of the back and front of the thorax, including the intercostals. The platysma always, and the face muscles and upper part of the trapezius usually escape. The legs also escape wasting as a rule.

Ocular and visceral symptoms are seldom observed.

Bulbar paralysis is a frequent complication. It is really due to an upward spread of the degenerative process in the cerebro-spinal axis.

Occasionally rheumatic-like pains may be complained of, but in the main the disease is an affection of purely motor elements, and the few pains are possibly due to overwork of muscles already enfeebled. Sensation is unaffected.

Though the disease ultimately involves both sides, it occasionally progresses in one hand for a considerable time before it appears in the other. Moreover, the condition may for a time assume the aspect of *subacute anterior poliomyelitis* with paralysis in excess of wasting, and with changes in the electrical reactions.

The symptoms which have been described are indicative of disease in the lower motor neurons. But occasionally the

disease of the upper neurons may be in advance of that in the lower, so that the affected muscles may be firm though wasted, and the tendon jerks may be preserved or even increased.

In the legs there is sometimes weakness without wasting (*spastic paraplegia*).

Diagnosis.—This is usually easy unless in an early stage when the wasting involves only a few muscles. The insidious onset, the progressive tendency, the place of commencement, the gradual extension to other muscles, the limitation to motor elements, and the age of the patient are the principal points.

In *syringomyelia* and *pachymeningitis*, there is sensory disturbance.

Idiopathic muscular atrophy is probable if the disease begins before twenty, and if other members of the family are affected.

In *lead palsy*, the paralysis usually takes the form of wrist-drop, but in some cases it closely resembles the disease now under consideration. The occupation of the patient, the presence of a lead-line on the gums, and a history of colic will make the diagnosis clear.

Prognosis.—The disease is usually progressive, but may become stationary, though often only at a late stage. Such patients, reduced to skin and bone, constitute some of the 'living skeletons' exhibited in museums. Bulbar paralysis is a common cause of death by interfering with deglutition and respiration. The latter is also hampered by the involvement of the intercostal muscles, and sometimes of the diaphragm. Respiratory diseases are in this way rendered very formidable, and food may get into the air-passages.

Treatment.—Mental and bodily fatigue and undue exposure should be avoided. Arsenic may be given internally, and massage and galvanism may also be tried. Otherwise the best treatment is that recommended by Gowers, viz., the hypodermic injection, once daily, at any place, of the nitrate of strychnine. The dose is $\frac{1}{50}$ of a grain to begin with, and should be increased up to $\frac{1}{20}$ or $\frac{1}{15}$. This measure must be persevered with for a year or two, with occasional short intermissions.

(3) Primary Spastic Paraplegia (Spastic Spinal Paralysis. Primary Lateral Sclerosis).

Definition.—A disease characterised by symptoms pointing to a primary degeneration of the upper motor neurons.

Etiology.—The disease generally begins in the first half of adult life. The sexes are almost equally liable. Concussion of the spine, repeated exposure, lead, gout, syphilis, influenza and other infections have been regarded as causes. A *family* or *hereditary group* may be distinguished in which the same symptoms occur in several members of a family and in several generations, both in young children and in adults. This variety resembles one form of cerebral diplegia except in so far as the decay of the neurons begins at puberty or later, instead of in infancy.

Morbid Anatomy.—This has not yet been satisfactorily demonstrated, and the lesion is largely a matter of inference. This is partly because the disease does not cause death, and partly because degeneration is rarely limited to the lateral columns. In one case of spastic paraplegia, no change was found in the cord after death. Gowers accordingly suggests that the degeneration begins in the terminations of the axons of the upper neurons in the grey matter of the cord. If it were limited to this part it could scarcely be detected. Erb, who first described the disease in 1875, still holds that the lesion is a grey degeneration of the pyramidal tracts, but he admits that the sclerosis is not always strictly confined to these tracts.

Symptoms.—The first symptom is weakness of the legs, which sets in very gradually. The limbs feel heavy and get tired too readily. Even for years, however, the patient may be able to walk considerable distances. The legs are stiff, and become in course of time the seat of 'clasp-knife rigidity,' through the severe extensor spasm at the knees. The knee-jerks are exaggerated and ankle clonus is present. The plantar reflex is extensor in type (Babinski's sign). The muscles are well nourished. The patient walks with his legs rigidly extended, and drags his feet on the ground. The

arms also in some cases suffer from weakness and rigidity. The cutaneous reflexes are exaggerated.

The cranial nerves, the sphincters, and sensation often escape altogether, but occasionally there are observed difficulty in swallowing and speaking, impaired action of the sphincters, and pains or numbness in the limbs or trunk. There are no trophic changes.

This disease is sometimes associated with general paralysis of the insane.

Diagnosis.—In a typical case the syndrome or symptom-group consists of four phenomena only : weakness of the legs, rigidity of the muscles, exaggeration of the tendon-reflexes, and the extensor type of plantar reflex. Genuine examples of the disease seem to be rare. Cases which correspond clinically to primary lateral sclerosis are apt to be found after death to be something else, such as insular sclerosis, old myelitis, amyotrophic lateral sclerosis, cerebral diplegia, or cerebral tumour.

It is important to keep separate from primary lateral sclerosis the *infantile* form of *spastic paralysis* which dates from childhood or from birth.

Spastic paraplegia may be *secondary* to myelitis, or to compression of the cord, but the history, mode of onset, examination of the spine, and associated symptoms should remove doubt.

In *ataxic paraplegia* there is inco-ordination.

Hysteria may give rise to spastic paralysis of the legs with exaggerated knee-jerks and even ankle clonus. But here the plantar reflex is likely to be normal or absent, and not extensor ; sensory disturbance may also be present ; the onset is rapid or even sudden ; and other evidences of hysteria may be present.

Prognosis.—There is practically no danger to life. The disease after a time may cease to progress. After the typical symptoms develop, the case may remain unchanged for a quarter of a century. In rare instances, practically all the muscles of the body become involved and the patient may die of exhaustion.

Treatment.—Complete rest should be tried for a time, and

over-exertion must always be avoided. Nerve tonics, such as arsenic and silver nitrate, with friction of the muscles, may be employed. Iodide of potassium and cod-liver oil have also been recommended. It may be desirable to give remedies for the purpose of lessening spasm ; among those which should have a trial are hyoscine hydrobromide, potassium bromide, and cannabis indica.

(4) Syphilitic Spinal Paralysis.

This disease was differentiated by Erb in 1892 from spastic spinal paralysis. It presents the typical clinical features of the latter disease, viz., the weakness of the legs, the muscular rigidity, the exaggeration of the tendon reflexes, and Babinski's sign, with other phenomena in addition. Firstly, it is almost confined to syphilitic subjects ; the symptoms may set in several years or many years after infection. At the same time injury, exposure, excesses, etc., may be contributory causes. Secondly, the function of the bladder is disturbed ; there is incontinence and sometimes retention of urine. Thirdly, there is a slight disturbance of sensation.

The onset is usually insidious, and the bladder trouble is frequently the first symptom.

Morbid Anatomy.—The lesion is a combined system disease, a primary, symmetrical sclerosis of several fibre systems in the cord. The tracts principally involved are the lateral pyramidal tracts, the direct cerebellar tracts, the tracts of Gowers, the columns of Goll and in part the columns of Burdach.

Diagnosis.—The disease is distinguished from *spastic spinal paralysis* (*primary lateral sclerosis*) by the affection of the bladder and the disturbance of sensation ; from *transverse myelitis* by the incompleteness of the motor and sensory paralysis, and by the absence of cystitis and bedsores ; and from *meningitis* by the absence of root symptoms.

Prognosis.—Recovery is likely to be incomplete at the best, but considerable improvement may take place. Some cases, however, die from acute or subacute exacerbations.

Treatment.—Erb recommends prolonged and energetic treatment with mercury and potassium iodide, but care

must be taken not to undermine the general health by such measures. Arsenic, strychnine and silver internally, with massage of the affected limbs, may also be employed.

(5) Ataxic Paraplegia (Combined Lateral and Posterior Sclerosis).

Etiology.—The disease is most common in males in the prime of life. Exposure, concussion of the spine and sexual excess are occasional causes. Syphilis is rarely, if ever, a cause. In many cases no cause can be recognised.

Morbid Anatomy.—There is sclerosis in the posterior and lateral columns. The lesion in the former accounts for the ataxy, and that in the latter for the paraplegia. The posterior sclerosis differs from that of locomotor ataxy first in not being specially marked in the lumbar region, and secondly in not being specially intense in the posterior root-zone. By these facts the preservation of the knee-jerk can be explained. Moreover, the lateral sclerosis is often not strictly confined to the pyramidal tract, but may extend further forwards and involve the dorsal and ventral cerebellar tracts, the direct pyramidal tract, and the antero-lateral efferent tract. The lesions, therefore, in this disease are not strictly systemic.

It is now known that many cases which correspond clinically to ataxic paraplegia are really examples of insular sclerosis. This fact may emerge through the development later on of symptoms which point definitely to insular sclerosis, or it may only be ascertained at the autopsy.

Symptoms.—The symptoms, as Gowers points out, are those of spastic paraplegia, plus inco-ordination. The legs suffer first, but the arms may be involved later on. Weakness and ataxy set in gradually. The ataxy becomes well marked and is apt to suggest tabes, but the deep reflexes, including the knee-jerk, are exaggerated.

Girdle sensation, pains in the legs, and loss of sensation are rare. The sphincters are sometimes involved.

Prognosis.—This is similar to that of spastic paraplegia. The disease scarcely tends to shorten life, but there is perhaps more danger from neglect of the bladder than in spastic

paralysis. Watch should always be kept for the appearance of nystagmus, intention-tremor, or involvement of the cranial nerves, since any of these would involve a revision of the diagnosis and prognosis.

Treatment.—This also resembles that of spastic paraplegia.

(6) Hereditary Ataxy (Friedreich's Ataxy. Hereditary Ataxic Paraplegia. Family Ataxy).

This is a form of ataxic paraplegia which differs from the ordinary form in occurring in several members of a family and in commencing at an early age. It is rarely inherited directly, and indeed its early onset usually makes marriage impossible ; but among the ancestors there may be a history of epilepsy, insanity, or some other neurosis. The two sexes are equally liable. The commencement is usually in childhood, sometimes in adolescence.

Morbid Anatomy.—There is sclerosis in the posterior, and to a less extent in the lateral and anterior columns. The posterior columns are affected from the lumbar region upwards as in tabes, but the posterior roots and Lissauer's tracts seem often to escape. The direct or dorsal cerebellar tract, and to a less extent the lateral pyramidal tract, are degenerated. There may also be disease of the cells of Clarke's column, the post-pyramidal nuclei and the hypoglossal nucleus. It is doubtful whether in this disease the overgrowth of interstitial tissue can be regarded as secondary (as it is in tabes) to the degeneration of the neurons. The two morbid processes may go on simultaneously as a consequence of defective developmental power in the neurons.

Symptoms.—The symptoms generally commence in childhood and without obvious cause. Weakness and unsteadiness gradually set in in the legs. The ataxy is not like that of locomotor ataxy, but more reeling, as in cerebellar disease and alcoholic intoxication. The patient keeps his legs apart as he walks. The unsteadiness is not always increased by closing the eyes. The weakness and inco-ordination gradually increase and extend to the upper limbs. The head and trunk become the seat of oscillating movements when the

patient sits up or attempts any movement. The knee-jerks are lost early. The plantar reflex is of extensor type.

Defective articulation from involvement of the tongue, nystagmus, especially on lateral deviation of the eyes, *pes cavus* or 'humped foot' (a form of club-foot with shortening of the foot and great hollowing of the instep), and lateral curvature of the spine are other symptoms. Of patients who have reached puberty, the males suffer from impotence, and the females from menstrual disturbance.

Marked sensory disturbance, disorder of the sphincters, pains, optic atrophy, iridoplegia, and mental disturbance are all absent as a rule.

Diagnosis.—*Locomotor ataxy* begins in adult life, and is characterised by pains and other disturbances of sensation, as well as by reflex iridoplegia, bladder troubles, and ataxic gait; while speech defects, nystagmus, oscillations of the head and spinal curvature are absent.

Insular sclerosis often occurs before adult life is reached, and may be associated with difficulties of speech, nystagmus, and oscillating movements of the trunk and head; but the knee-jerks are usually exaggerated, mental disturbance is common, the gait is spastic, and there is no deformity of the foot or spine. The occurrence of more than one case in a family is strong evidence in favour of Friedreich's disease.

Prognosis.—The disease is progressive and incurable, but has little tendency to cause death.

Treatment.—No remedy is known.

(7) Hereditary Cerebellar Ataxy.

This is a disease which presents symptoms akin to those of Friedreich's ataxy, but differs from it in the following respects. It generally begins after twenty years of age, the knee-jerks are exaggerated, and optic atrophy is common. Atrophy of the cerebellum has occasionally been found and is supposed to account for the symptoms.

(8) Scleroses connected with Morbid Blood-states.

In certain forms of grain poisoning (ergotism, lathyrism and pellagra), there may be simple or combined system degenerations in the cord. Diabetes is occasionally associated with posterior sclerosis. In pernicious anæmia, there may be changes in the posterior and lateral columns with sensory and motor disturbances in the legs. As either the anæmia or the cord symptoms may lead the way, it is possible that both are due to the same poison.

(9) Subacute Combined Degeneration of the Spinal Cord.

This name has been given to a disease which, so far as the nervous phenomena are concerned, might well be regarded as chronic myelitis; but severe anæmia and cachexia are present in addition. It was formerly supposed that the anæmia was the cause of the cord lesion, but it is more likely that the two are due to the same cause. The disease is chiefly seen in middle life, and includes among its possible causes syphilis, alcohol, prolonged suppuration, and parturition.

Morbid Anatomy.—The principal changes are in the dorsal region of the cord, where there is a diffuse degeneration of the white matter. Secondary ascending and descending degenerations are present above and below this lesion. The grey matter escapes.

Symptoms.—In the first stage there is slight spastic paraplegia with ataxy and sensory disturbances, though the patient is still able to go about. This may pass abruptly into the second stage, when walking and standing become impossible, anæsthesia becomes marked, and girdle sensation, severe pains in the legs, ankle clonus, Babinski's sign and irregular pyrexia may be observed. This, again, passes abruptly into the third stage, which is characterised by complete flaccid paralysis, muscular hypotonus, loss of the knee-jerks, incontinence of urine and fæces, and complete anæsthesia which frequently reaches up to the first dorsal

level. Fever, delirium, sacral bedsore, œdema and cachexia are phenomena of this stage. The upper limbs may suffer to some extent, but the neck and cranial nerves usually escape. The anæmia may resemble that of pernicious anæmia, but is generally of the secondary type.

Prognosis.—The disease terminates fatally in the course of a period varying from three months to three years.

Treatment.—Iron and arsenic may be helpful for the anæmia and cachexia, but so far as is known no medicine is of any use for the disease in the cord.

DISEASES OF THE MEDULLA OBLONGATA

i. Chronic Bulbar Paralysis

(Labio-glosso-laryngeal Paralysis. Labio-glosso-pharyngeal Paralysis).

Etiology.—Little is known as to the cause of this disease. It attacks males more frequently than females. It almost never occurs before forty, and is thus more a senile disease than progressive muscular atrophy, which is the corresponding affection of the cord.

Morbid Anatomy.—The medulla oblongata is generally normal to the naked eye. In one considerable group of cases, the fibres of the affected muscles are atrophied or degenerated. This depends on degeneration of the lower motor neurons connected with these muscles, viz., the cells in the motor nuclei and the corresponding motor nerve fibres. The hypoglossal nucleus innervates the tongue, and apparently (by fibres distributed through the facial nerve) the orbicularis oris. The medullary part of the spinal accessory innervates the palate and larynx, and the ninth or tenth nerve supplies the pharynx. The fibres passing from the motor area of the cortex to the bulbar nuclei may also be wasted, in which case both upper and lower neurons are degenerated as in most cases of progressive muscular atrophy.

In other cases of this paralysis, however, the muscles are not wasted, and the condition is then supposed to depend on

degeneration of the upper neurons only, as in primary spastic paraplegia.

The changes connected with progressive muscular atrophy are present in many cases in addition to the bulbar changes.

Symptoms.—The first symptom is usually a defect in articulation which results from weakness of the tongue, and is often attributed to some dental defect or operation. For a time, however, the tongue can still be protruded. It is sometimes large and soft, in other cases wasted and wrinkled. Then the lips become weak and cannot be properly brought together. They may be normal in size or wasted. The patient cannot whistle, and his pronunciation is further interfered with. It is important, however, to remember that the deliberate efforts made during the course of an examination may not betray defects of speech to nearly the same extent as ordinary conversation. The saliva can neither be swallowed nor retained, so that it constantly dribbles from the mouth, and the patient goes about with a handkerchief in his hand. Weakness of the palate causes a nasal tone of voice, and is a third agent which impairs articulation. Swallowing becomes difficult through weakness of the tongue, palate and pharynx. The weakness of the tongue increases till the organ cannot be protruded and mastication is difficult. The laryngeal weakness impairs phonation and cough, and the speech becomes less and less intelligible. The nasolabial furrows become very marked owing to contracture of the zygomatic muscles, which seem to suffer less than their opponents.

Sensation and taste are unaffected.

Reflex action fails in the palate, fauces, and even the larynx, as a result of the damage to the motor structures.

Electrical irritability is not much altered, as a rule.

Not unfrequently the patient is emotional to an abnormal degree.

Bulbar palsy and progressive muscular atrophy often coexist. Either may set in before the other.

Diagnosis.—This depends on the peculiar distribution, symmetrical character, gradual onset and progressive tendency of the paralysis. A *tumour* in or near the medulla is

likely to cause sensory symptoms and head symptoms, and the palsy may be unilateral for a time. *Chronic pseudo-bulbar paralysis* (due to lesions of both hemispheres) is rare. The symptoms are likely to develop in one side before the other, and to be associated with weakness of the limbs.

Prognosis.—This is very serious. Death is usually due to exhaustion by starvation, but it may result from disease of the respiratory system.

Treatment.—Strychnine should be given hypodermically as in progressive muscular atrophy ($\frac{1}{50}$ grain gradually increased to $\frac{1}{15}$ grain). The patient should speak as little as possible, and must take great care in feeding. In advanced stages it may be desirable to teach him the use of the stomach tube, so that the risk of food entering the larynx may be diminished.

ii. Other Forms of Bulbar Paralysis.

Apoplectiform Bulbar Paralysis has a distribution similar to that of the chronic disease, but its onset is sudden. It is due to a vascular lesion—generally thrombosis of one or both vertebral arteries, such as occurs in atheroma and syphilis.

Symptoms.—The onset is sudden, but headache and giddiness frequently precede the actual attack. Consciousness is rarely lost. The power of moving the tongue, of speaking, and of swallowing is suddenly impaired ; and there may also be cough, dyspnœa, and weakness or convulsions in the limbs from irritation of, or pressure on neighbouring nerve centres and tracts. The distribution of the paralysis is not always so symmetrical or regular as in the chronic disease.

Diagnosis.—The chief difficulty is the diagnosis from the pseudo-bulbar paralysis which results from lesions of both hemispheres. In the latter case, however, the two sides are likely to suffer, not simultaneously, but one after the other, and hemiplegic symptoms will be more marked than in genuine bulbar paralysis.

Prognosis.—Death often takes place within a few hours, but the patient may survive with partial paralysis, which may or may not be associated with wasting.

Treatment.—If syphilitic vascular disease is suspected, specific treatment must be promptly enforced. Otherwise the treatment must be general and symptomatic.

Acute Inflammatory Bulbar Paralysis (*myelitis bulbi, acute inferior polioencephalitis*) is an acute inflammation of the grey matter of the bulb, analogous to acute anterior poliomyelitis in the cord, and to acute inflammation of the nuclei of the motor ocular nerves (superior polioencephalitis) higher up in the brain. It is a rare disease.

Pseudo-bulbar Paralysis is generally due to a sudden vascular lesion involving the motor area of the cortex on both sides. Much less frequently the lesions are in the region of the internal capsules. The most common cause is thrombosis in connection with disease of the vessels. The condition may usually be distinguished from bulbar paralysis by the fact that one side suffers after the other, and by the presence of hemiplegia at the onset.

iii. Myasthenia

(Myasthenia Gravis. Myasthenia Bulbaris. Asthenic Bulbar Paralysis).

This is a rare disease which affects young adults of either sex. No anatomical change has been discovered which could account for the symptoms, though in a few instances disease has been found in the thymus. It has been supposed that a toxin, either developed in the body, or introduced from without, poisons some portion of the motor apparatus, but the evidence for this view is far from complete. In a few cases the disease has followed an acute infection, chill, emotion, or exertion.

Symptoms.—The muscles are feeble and are rapidly exhausted by use, but on resting quickly regain such power as they possess. The *myasthenic reaction*, which is often present, is an illustration of this feature : a faradic current sufficient to cause tetanus of the affected muscles soon loses its effect, so that the muscles cease to contract, but after a short period of rest, they are found to be again capable of responding to the current. Some muscles, however, are the

seat of more or less permanent weakness. There is no muscular wasting except in rare cases, and there is no change in the response of either nerves or muscles to galvanism.

Though the disease involves the muscles generally, those which are innervated from the medulla oblongata are specially apt to suffer. Thus the lips, palate and pharynx, and also the masticatory and external ocular muscles are commonly involved. There may be ptosis and imperfect movement of the eyeballs ; indeed, ptosis is often the first sign of weakness. Gowers has called attention to an alteration in the smile (*nasal smile*) which may result from weakness of the zygomatic muscles ; the angle of the mouth is not moved outwards, and the action of the levators produces a furrow which is entirely above the upper lip. Involvement of the masticatory muscles may compel the patient to rest two or three times in the course of a meal. Weakness of the lip muscles may abolish the power of whistling and impair articulation. Weakness of the palate may induce a nasal tone of voice, and even permit of regurgitation of liquid foods through the nose. Protrusion of the tongue may be impossible. Weakness of the respiratory muscles may be a grave feature of the case. Deglutition is almost always involved, and the muscles of the neck, trunk and limbs are sometimes at fault. The knee-jerks, though normal at first, may quickly diminish and almost disappear on repetition, but after a short rest they become as active as ever. The symptoms are not always strictly symmetrical.

Sensation and the sphincters are unaffected. The pupils and the larynx also escape as a rule.

The symptoms vary much from time to time, and are aggravated by excitement, exposure, menstruation, etc.

Prognosis.—About a third of the recorded cases have died, usually after a year or two, and usually from asphyxia or some pulmonary trouble. But life may be prolonged for many years, and recovery may take place. The weakness may vary very greatly from time to time.

Diagnosis.—Mild myasthenia might be mistaken for neurasthenia, but if the myasthenic reaction is present, or if

any permanent paralysis is present, simple neurasthenia can be excluded.

Treatment.—Rest and avoidance of chill and excitement are indicated. Artificial feeding and even artificial respiration may be necessary. The hypodermic injection of strychnine has occasionally proved beneficial. Iron, arsenic, and massage may also be tried.

DISEASES OF THE BRAIN.

i. Introduction.

Cerebral Anatomy and Localisation.

The *motor area* of the cortex, containing the upper ends of the upper neurons, was until recently supposed to occupy the ascending parietal as well as the ascending frontal convolution. But recent investigations by different methods, including experiments on the cortex of anthropoid apes, studies in the histology of the human cortex in health and in amyotrophic lateral sclerosis, and electrical stimulation of the cortex in cases of operation on the human brain, are generally accepted as proving that the motor area does not extend behind the Rolandic fissure (central sulcus). Practically speaking, the motor area in man corresponds to the ascending frontal convolution (precentral gyrus). The leg area is at the upper end of the convolution, the arm area lower down but rather above the middle, and the face area still lower. The trunk is represented between the leg and arm centres, the neck between the arm and face centres, and the tongue between the face centre and the Sylvian fissure. Irritative disease of the motor area causes convulsions, beginning in the muscles corresponding to the part irritated; destructive disease causes paralysis of the corresponding muscles.

The motor area appears to have some sensory function in addition, but there is much uncertainty as to its nature and extent. Damage to this area may cause impairment of sensation, chiefly at the extremity of the affected limb, but it does not cause complete anæsthesia. Similarly a con-

vulsion beginning in a limb may be associated with, or preceded by sensory disturbance—*e.g.*, tingling.

The functions of the *prefrontal lobe* are not well understood. Broca's centre in the third (left) frontal convolution will be alluded to in connection with aphasia. The centre for deviation of the head and eyes to the opposite side is supposed to exist higher up in the prefrontal lobe, and if this lobe is affected, especially by bilateral disease, the mental functions are apt to be impaired.

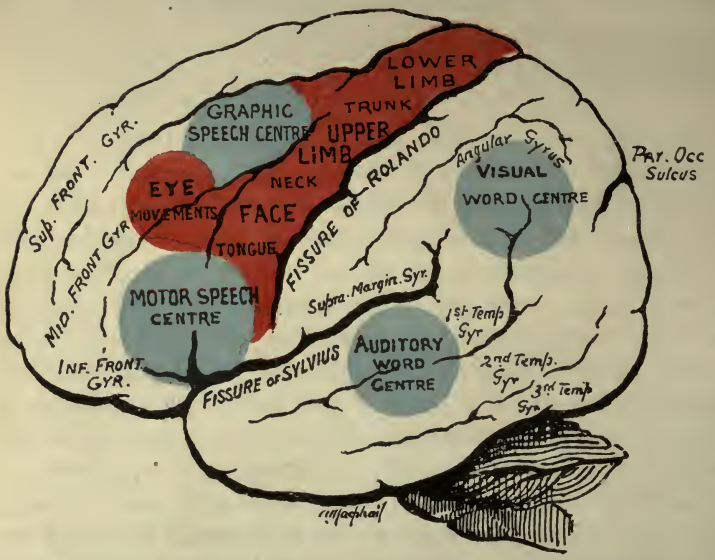

FIG. 39.—LATERAL ASPECT OF LEFT CEREBRAL HEMISPHERE.
The so-called motor area is coloured red. The four speech centres are in blue.

The *ascending parietal convolution* or *postcentral gyrus* appears to contain the terminations of the axons of the third order of neurons of which the main sensory path is composed (p. 624). The ascending parietal convolution, therefore, is connected with sensation in the opposite limbs. The *angular region* or posterior portion of the inferior parietal lobule is the highest in type of the *visual centres*. It is connected with the occipital lobe, both of its own and of the other side, and in this way is connected with the whole of both retinæ. Destruction of one angular gyrus is sup-

posed to cause *crossed amblyopia*, namely, general impairment of vision in the opposite eye, with concentric diminution of the visual field. Vision is affected in both these ways on the side of lesion also, but to a much less extent than on the opposite side. Destruction of the angular region on the left side causes one form of *aphasia*.

The *occipital lobe*, particularly in the region of the calcarine fissure (Fig. 40), represents the corresponding half of each retina. Thus destruction of the left calcarine region causes blindness of the left halves of both retinæ, so that the patient cannot see to his right. The same result (*lateral*

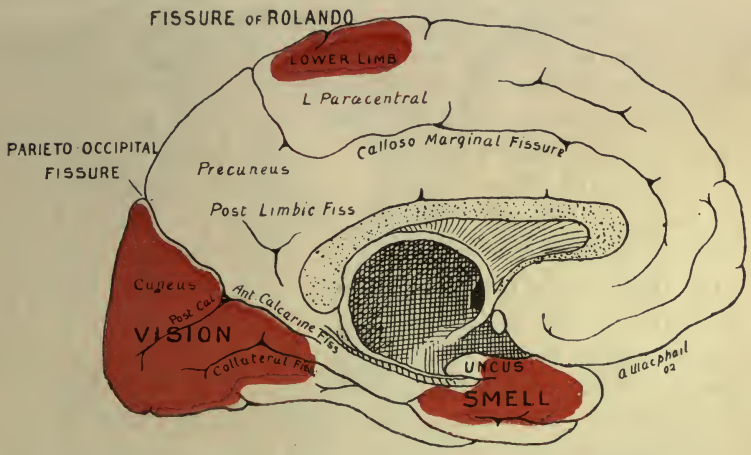

FIG. 40.—MESIAL ASPECT OF LEFT CEREBRAL HEMISPHERE.
Some of the cortical centres are coloured red.

hemianopia) follows destruction of the optic radiations and of the primary optic ganglia, as mentioned in connection with affections of the optic nerve.

The *auditory centre* is situated in the posterior part of the first convolution of the *temporal lobe*. It is connected with both ears, but especially with that on the opposite side. Destruction causes temporary deafness in the opposite ear. Bilateral destruction naturally causes complete deafness. Irritation, as by a tumour, may cause subjective noises. Disease on the left side causes one form of *aphasia*.

The *uncinate gyrus* and perhaps the hippocampal gyrus

on the mesial aspect of the hemisphere are related to the sense of *smell*. The *gustatory* centre is believed to be situated in close proximity to the olfactory centre.

Disease of the *centrum semi-ovale* or *corona radiata* gives rise to symptoms resembling those of cortical disease if the lesion is close to the cortex, and to symptoms like those of disease of the internal capsule if the lesion is near the capsule.

The *corpus callosum* is a system of commissural fibres uniting centres in the two hemispheres. The symptoms which have been observed in disease of this part are in no way characteristic.

The *internal capsule*, as shown by the accompanying figure (Fig. 41), consists of an anterior and posterior limb, the former separating the lenticular nucleus from the caudate nucleus, and the latter separating the lenticular nucleus from the optic thalamus. At the *genu* or bend of the capsule, where the two limbs meet, there run the upper neurons of the motor path from the cortex to the muscles of the face and tongue. Behind these, viz., in the anterior third of the posterior limb of the capsule, there run the upper neurons of the path to the muscles of the upper limb. Still further back, viz., in the middle third of the posterior limb of the capsule, there are the upper neurons of the path to the muscles of the lower limb. Hæmorrhage in the region of the capsule is one of the commonest causes of *hemiplegia*.

The posterior third of the posterior limb contains sensory tracts, so that a lesion here may cause *hemianæsthesia* on the opposite side of the body. In the same region there are tracts connected with the special senses, and for this reason Charcot called it the *sensory crossway* (the visual defect caused by a lesion in the crossway is lateral hemianopia). Irritation of sensory fibres in this region may cause pains in the limbs.

Extensive destruction of the *caudate* and *lenticular nuclei* may apparently take place without causing either motor or sensory symptoms, though these may follow if the lesion should extend to, or cause compression of, the neighbouring internal capsule.

Disease of the *optic thalamus* may cause athetoid movements in the opposite hand, or it may cause loss of emotional

movement, with preservation of voluntary movement, in the opposite side of the face. Hemianopia may result from disease of the *pulvinar* or posterior part of the thalamus.

Disease of the *corpora quadrigemina* is to be suspected in a case of cerebral tumour associated with a reeling gait like that of cerebellar disease, and with bilateral ophthalmoplegia not affecting both eyes or all the ocular muscles to the same degree. The knee-jerks may be lost or exaggerated.

In the *crus cerebri*, the upper neurons of the motor path

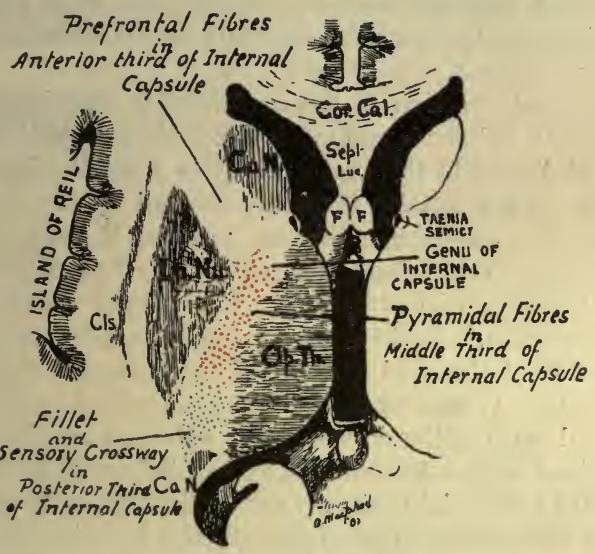
Prefrontal Fibres in Anterior third of Internal Capsule
Cor. Cal.
Sept. Lue.
ISLAND OF REIL
F F
TAENIA SEMICI
Genu of INTERNAL CAPSULE
Cls
Pyramidal Fibres in Middle Third of Internal Capsule
Fillet and Sensory Crossway in Posterior Third of Internal Capsule

FIG. 41.—HORIZONTAL SECTION OF PART OF THE BRAIN, SHOWING THE INTERNAL CAPSULE AND ITS RELATIONS.

are situated in the middle of the ventral or crustal portion. Some of them cross the middle line in the crus to reach the opposite oculomotor nucleus. The sensory neurons are in the dorsal or tegmental portion of the crus. A characteristic result of disease in one crus is hemiplegia involving the leg, arm and lower face of the opposite side, and the third nerve on the side of lesion (one form of *crossed paralysis*).[1] If the lesion involves the tegmentum, sensation may be

[1] The form of crossed paralysis just described as resulting from a lesion in the crus cerebri is sometimes spoken of as *Weber's syndrome*.

affected. Occasionally a tumour involves the crus and optic tract together.

Lesions in the *pons* may be unilateral or bilateral. The former often causes a *crossed paralysis* characterised by hemiplegia involving the opposite limbs, together with palsy of the fifth, sixth or seventh nerve on the side of lesion. Conjugate movement of the eyes to the side of lesion may be lost. Bilateral disease causes bilateral paralysis, and if acute, may cause rigidity of the limbs, contraction of the pupils and hyperpyrexia.

Lesions of the *medulla oblongata* are considered under Bulbar Paralysis.

The great function of the *cerebellum* is co-ordination of movement. There is still much uncertainty as to the symptoms of disease in this organ, and this is largely due to the fact that compensation is readily effected by the surviving tissue. Thus in chronic disease there may be no distinctive symptoms. The principal symptom of cerebellar disease is disturbance of equilibrium. The patient walks like a drunken man. Lesions of the middle lobe may cause retraction of the head, arching of the back and rigidity of the limbs. Weakness of the trunk muscles has also been ascribed to cerebellar disease. The knee-jerk may be altered in various ways. Pressure by a one-sided cerebellar tumour upon the pyramidal tract may cause weakness in the opposite limbs, with exaggeration of the opposite knee-jerk, but as a rule the weakness and tendency to fall are on the side of lesion. Such a tumour may paralyse cranial nerves (*e.g.*, the fifth, sixth, seventh or eighth) on the side of lesion. Nystagmus and intention-tremor are other symptoms. Tumour and abscess commonly cause vertigo, stupor, vomiting and severe optic neuritis. Hydrocephalus may develop in children from obstruction of the veins of Galen. Tumour is by far the most common lesion met with in the cerebellum.

The Cerebral Blood-supply.

The motor area of the cortex is supplied by the *middle cerebral* artery, which also supplies the auditory region of the cortex and the greater part of the angular or highest visual

region. Thus all forms of permanent aphasia are referable to lesions in the territory of the middle cerebral artery.

Shortly after its origin, the middle cerebral gives off a number of fine arteries which pass into the anterior perforated space to supply the internal capsule, lenticular nucleus, and parts of the caudate nucleus and optic thalamus. Some of these small central arteries pass upwards for a little distance outside the lenticular nucleus, and then pass through its outer segment and through the capsule ; those in front reach the caudate nucleus (*lenticulo-striate*), and those behind reach the optic thalamus (*lenticulo-optic*). These arteries are very apt to rupture, and one of the lenticulo-striate group was named by Charcot the *artery of cerebral hæmorrhage*.

The cortex of the occipital lobe is supplied by the *posterior cerebral* artery, which also supplies the crus and the primary optic ganglia (thalamus, and geniculate and quadrigeminal bodies). This vessel may contribute to supply the angular region.

The cerebellum, pons and bulb are supplied by the *vertebral* and *basilar* arteries.

The sluggish character of the circulation in the *cerebral sinuses* should be borne in mind. Not only have the veins on the surface of the hemisphere an ascending course, but they open forwards into—*i.e.*, against the current in—the superior longitudinal sinus. Thrombosis is thus intelligible.

Communicating vessels pass through the skull between the sinuses inside and the veins outside, so that disease inside may cause venous distension or œdema outside.

ii. Cerebral Meningitis.

Several varieties of meningitis, including the *epidemic* (p. 76), *posterior basic* (p. 80), *tubercular* (p. 108) and *syphilitic* (p. 149), have been described in Section I. in connection with the infections. Of those which must now be considered, some involve the dura mater (*pachymeningitis*), and others the soft membranes or pia-arachnoid (*lepto-meningitis*). Moreover, inflammation may affect either the outer or the inner layer of the dura.

External Pachymeningitis is generally due to injury or disease of the neighbouring bone, and hence belongs to the province of the surgeon.

Internal Pachymeningitis is chiefly observed in its *hæmorrhagic* form, constituting the *hæmatoma of the dura mater* (*subdural false membrane, subdural deposit, meningeal blood tumour*). It occurs most commonly in males, and in the later period of life. It is usually associated with general paralysis or some other chronic insanity, or with chronic alcoholism ; but it sometimes occurs in acute fevers and hæmorrhagic diseases. The condition is rarely seen outside asylums.

Morbid Anatomy.—The lesion may be unilateral or bilateral. Between the dura mater and arachnoid there is a series of layers of membranous-looking tissue, and between these layers there are collections of blood or blood-clot, or organised clot.

Pathology.—One view is that the hæmorrhage is primary, another that inflammation comes first. In a case examined by myself at the Victoria Infirmary, hæmorrhage appeared to have been the primary fact. The disease is described as a form of meningitis because Virchow, half a century ago, advocated this view. But different writers before Virchow had considered it to be primarily hæmorrhagic, and this theory has been largely held by British alienists. Atrophy of the brain tissue, especially if the dura mater is degenerated and congested, will favour subdural hæmorrhage of a compensatory kind. The epileptiform seizures of general paralysis and certain other kinds of insanity favour the rupture of degenerated vessels by causing serious changes in intracranial pressure. In sane and otherwise healthy persons, meningeal hæmorrhage resulting from injury is not likely to cause a subdural hæmatoma, because a small quantity of effused blood will soon be removed by the normal agencies, and a large effusion will cause pressure symptoms and lead to death if it is not removed by operation. At the same time, in the insane, a slight injury to the head may be the exciting cause of a hæmorrhage which produces a hæmatoma.

Symptoms.—There may be no symptoms, and symptoms when present are not distinctive. They include headache, apoplectiform seizures, convulsions and hemiplegia. In the case already mentioned, the patient was suddenly seized in the midst of apparent health with convulsions which were at first general and afterwards right-sided. Death took place in eighteen hours, and an old-standing hæmatoma was found over the left side of the brain. In another case the patient was a woman who had suffered from permanent amaurosis of sudden onset, optic atrophy and epilepsy. Death was apparently due to granular kidney, and the autopsy revealed a large porencephalus in addition to the hæmorrhagic pachymeningitis.[1]

Diagnosis.—Internal hæmorrhagic pachymeningitis is rarely diagnosed during life. The other symptoms present in the case mask those due to the development of the hæmatoma.

Prognosis.—The subdural hæmorrhage is seldom the direct cause of death. The patient usually dies of the primary disease or of some intercurrent affection. A severe hæmorrhage, however, may cause death with apoplectic symptoms.

Treatment.—This includes rest, elevation of the head, cold applications, and gentle purgation.

SUPPURATIVE LEPTOMENINGITIS is most common as a complication of pneumonia, empyema, or bone disease in connection with chronic otitis media. It may also complicate or follow erysipelas, pyæmia, infective endocarditis, scarlet fever, small-pox, rheumatism, and other acute diseases. It may be due to extension from disease of the nose, or syphilitic disease of the cranium. It may follow injuries of the head.

The most common microbe is the pneumococcus. In erysipelas the streptococcus is common. In many cases the infection is mixed.

Morbid Anatomy.—In the early stage there is increased vascularity of the membranes, which contain an excess of watery fluid. Later on, yellow exudation, and still later, greenish-yellow pus cover more or less of the brain surface.

[1] *Journal of Mental Science*, July, 1904.

The exudation is often most marked over the convexity, but is frequently present also at the base of the brain, and in the spinal membranes. It follows the pia mater into the sulci of the cortex. The underlying brain tissue is often softened and may contain small foci of suppuration (*meningo-encephalitis*). In most cases some lesion will be found to which the meningeal inflammation is secondary.

Symptoms.—When meningitis sets in in the course of an acute fever, there may be no definite change in the symptoms to indicate what has taken place, and this is particularly the case when the inflammation involves the convexity and spares the base and the cranial nerves. When symptoms occur, the most important are headache, vomiting, optic neuritis, delirium, convulsions, paralysis of cranial nerves and of the limbs, constipation, and disturbances of the pulse, respiration and temperature.

Headache is one of the most constant and early symptoms, and is generally an important feature all through the illness, at least as long as consciousness is preserved. Even when the patient is only partly conscious, he frequently gives utterance to a sudden scream of agony, the *hydrocephalic cry*,[1] which is elicited by an exacerbation of the pain. In exceptional instances, headache is practically absent. Vomiting is another common and early symptom. It is apt to occur without obvious exciting cause, and may be unaccompanied by nausea.

Optic neuritis is seen chiefly when the meningitis is at the base of the brain. It does not occur till the inflammation of the membranes has been present for some time, and it is never so severe as it frequently is in connection with cerebral tumour.

Delirium is common and assumes varying aspects. As Gowers points out, its diagnostic importance lies in the fact that it accompanies the headache, instead of—as in general febrile conditions—taking its place.

Convulsions are not uncommon, especially in children. Tonic spasm of the muscles at the back of the neck causing

[1] This cry may occur also in acromegaly with tumour of the pituitary body, and in other forms of cerebral tumour.

retraction of the head, and similar rigidity of the muscles lower down the trunk, constitute important evidence of spinal meningitis at the corresponding levels.

The pupils are often contracted in the early stage, and dilated in the final stages of the disease. Irritation of, or damage to the cranial nerves may also give rise to inequality of the pupils, strabismus, ptosis, paresis of the face and deviation of the tongue. Symptoms referable to the cranial nerves are naturally due chiefly to meningitis at the base, but inequality of the pupils may be due to disease at the convexity.

The limbs are sometimes rigid or weakened on one or both sides.

There is sometimes abnormal sensitiveness of the skin, and of the senses of sight and hearing, so that light and noise increase the patient's distress.

Fever is present, but is irregular in type. The pulse is sometimes rapid, sometimes abnormally slow ; it frequently fails to keep pace with the temperature. Cheyne-Stokes respiration or some other abnormal type of breathing may be present (see p. 259). The *tache cérébrale* may be well marked and very persistent. The bowels are constipated, and the abdomen is retracted. In the final comatose stage, the evacuations are passed into the bed.

When distinctive symptoms are present in this form of meningitis, they are commonly ushered in abruptly with rigors and high fever ; and they generally end in death within two or three days.

Diagnosis.—In *cerebral hæmorrhage*, the symptoms usually set in much more quickly than in even the most acute forms of meningitis.

An *intracranial tumour* generally runs a chronic course, and is often associated with severe optic neuritis. The existence of a possible cause of meningitis is important—*e.g.*, pneumonia, pyæmia, or some other infection, ear disease, injury to the skull, etc.

In *acute fevers*, delirium replaces the headache instead of being added to it. Palsies of cranial nerves and other localising evidence of intracranial disease are of great

significance. Kernig's sign suggests that the spinal membranes are involved, but it is not conclusive evidence of meningitis, since it has been observed in various other diseases such as enteric fever, uræmia and sciatica.

Cerebral abscess has less tendency than meningitis to involve the cranial nerves. Moreover, the early symptoms are generally of much longer duration than in suppurative meningitis. But the diagnosis may be very difficult, since the two conditions may result from similar causes—*e.g.*, disease and injury of the bones of the head—and may, moreover, coexist.

The rapid course of suppurative meningitis differentiates it from most cases of *tubercular* and *posterior basic meningitis*. The existence of a possible cause is also of service in diagnosis.

Prognosis.—The outlook is almost hopeless.

Treatment.—Prophylactic measures are naturally of great importance. Once meningitis has set in, it might still be justifiable to try to remove the primary lesion (*e.g.*, disease in the ear, thorax, etc.). Ice-bags and leeches may be applied to the head. Large doses of the tincture of the perchloride of iron may be given internally. If the particular organism which is the cause of the disease can be discovered by lumbar puncture or by examination of the blood, or is indicated by the presence of some infection such as pneumonia or erysipelas, the appropriate serum should be employed. Lumbar puncture may be used as a therapeutic agent.

iii. General Paralysis of the Insane

(General Paralysis or Paresis. Paralytic or Paretic Dementia. Chronic Diffuse Meningo-encephalitis).

Definition.—A chronic progressive disease of the cerebral cortex associated with both mental and motor enfeeblement, characterised by a special defect in articulation and often by delusions of grandeur, spreading to other parts of the nervous system, and ending fatally within a few years.

Etiology.—The great majority of the patients have had syphilis; general paralysis, like tabes dorsalis, is a parasyphilitic affection. The disease is more common in men than in women. In women it is especially common among prostitutes. It attacks men in the prime of life, at the time when they ought to be the most useful members of society. *Juvenile general paralysis* is a rare form which sets in about puberty (it may be as early as the ninth year, or as late as the twenty-fourth), and attacks males and females in almost equal proportions. It is almost invariably due to inherited syphilis, though occasionally perhaps to the acquired disease. Alcoholism, city life, constant mental strain and sunstroke may be mentioned as additional causes. Ford Robertson regards the disease as a specific infection due to an organism resembling the diphtheria bacillus, which usually enters the body by way of the digestive and respiratory tracts.

Morbid Anatomy.—The calvarium is thickened and the diploe is obliterated. The dura mater is thickened and firmly adherent to the skull-cap. Hæmatoma of the dura mater (hæmorrhagic internal pachymeningitis) is sometimes present. The soft membranes are thickened and present white patches. The pia mater is so adherent to the tops of the convolutions, especially in the anterior and central regions, that in removing it the cortex is torn, and the latter then presents a mouse-eaten appearance. The brain as a whole is reduced in size, and the convolutions, especially in the frontal and parietal regions, are wasted. Where the convolutions are wasted, there is an excess of fluid in the soft membranes. The brain tissue is soft, and hyperæmia and anæmia are distributed irregularly in the grey matter. The ventricles of the brain contain an excess of fluid and their lining membrane is granular.

Microscopic investigation shows all stages of degeneration in the nerve cells of the cortex. The tangential and radial nerve fibres in the cortex also undergo degeneration. The walls of the fine arteries are thickened and degenerated, and leucocytes accumulate in the perivascular sheaths. The connective tissue elements are increased, especially in the superficial layer of the cortex.

Changes analogous to those which have been described involve practically the whole nervous system, both central and peripheral. The membranes of the cord may resemble those of the brain. The cord itself may be shrunken and soft. The cells of the anterior horns undergo degeneration, and the posterior and lateral columns are frequently the seat of sclerosis.

Pathology.—Two principal theories of the disease are held. One view is that the primary lesion is inflammatory, a meningo-encephalitis commencing in the sheaths of the arterioles and causing secondary degeneration of the nervous elements. According to the other theory, the degeneration of the neurons is primary, and the other changes are secondary. Mott, who holds the view that the primary condition is degeneration of nervous elements, thinks that the secondary local changes, as well as some of the symptoms, are due to local irritation and general intoxication by the products of degeneration.

Symptoms.—The first symptoms noted are in some cases mental and in others motor. As the patient is often an active business man, a very slight change may be of great significance. Changes of character, irritability, forgetfulness, inattention to business, abnormal facility, and offences against propriety may be early mental symptoms. Delusions of grandeur are common, the patient fancying that he is immensely wealthy or strong or exalted in station, and often acting on such belief. He may be violent, noisy, sleepless and homicidal. Melancholia is less common. In cases with depression, there may be delusions of perdition, persecution, etc. The patient may refuse food, and attempt to commit suicide. In a third group, excitement and depression alternate with one another. In a fourth group, which includes many cases, there is progressive mental failure from the outset, without much in the way of either exaltation or depression. The memory, will-power, and judgment fail. The patient becomes childish and untidy, and loses control over the sphincters.

Among the motor symptoms, there are tremor of the tongue and lips, a slurring articulation, lack of facial ex-

pression, inequality of the pupils, the Argyll-Robertson pupil, and weakness of the legs with exaggerated knee-jerks, or ataxy with loss of the knee-jerks. Epileptic seizures may occur early. The defect of articulation is particularly noticeable when the patient attempts to pronounce long words containing the letter *r*. The letters which he writes reveal in a striking manner the disorder of the intellect, and the tremor and weakness of the muscles.

The period of the disease which elapses before chronic mental failure is definitely established, and which is characterised by the early symptoms already mentioned, is sometimes described as the *first stage*.

In the *second stage*, the chronic dementia may be marked chiefly by apathy and calmness, though sometimes there is excitement or depression. The grandiose delusions may now be well marked. They seem to occur especially among well-educated people, and are often absent in patients of the humbler classes. The memory, will, and reasoning power are greatly impaired. On the physical side, there may be blunting of the senses of smell, taste and sight, as well as touch. The patient may take quinine with as much relish as sugar. The optic discs are sometimes atrophied. Injuries may be sustained and overlooked. A hæmatoma often develops in the ear. Control over the sphincters is lost. There is slight elevation of temperature, and the patient loses flesh. Apoplectiform, epileptiform, and other alarming attacks may occur, and each, if the patient survives it, tends to leave the mental condition more degraded than before.

In the *third stage*, there is complete paralysis of mind and body, and the patient lies in bed unable to attend to himself in any way. Motion and sensation, emotion and will are gone. Bedsores and other trophic lesions readily develop. Death may be due to asthenia, coma, convulsions, or some visceral complication.

Diagnosis.—The important point is the combination of mental and motor symptoms, the latter including the tremor of the lips and tongue and the impairment of articulation. The last is often very noticeable when the patient tries to pronounce words containing the letters *r* and *l*, such as

'truly,' 'rural,' 'artillery,' etc. As long as the symptoms are purely motor or purely mental, there is room for doubt, though a strong suspicion may often be entertained. Some cases present for a time the aspect of tabes dorsalis, spastic paralysis or epilepsy. The diagnosis is cleared up by the subsequent course of the disease. Some cases, however, must be regarded as a combination of tabes and general paralysis (*tabo-paralysis*). Either the mental or the spinal symptoms may be first observed, or both may be present from the outset.

Cerebral syphilis is sometimes very like general paralysis, but paralysis of the limbs and cranial nerves usually sets in early, and there is not likely to be the same disorder of speech, tremor of the tongue, or delusion of grandeur.

Lead poisoning and *chronic alcoholism* may give rise to symptoms very similar to those of general paralysis. In the former there may be a lead-line on the gums, wrist-drop, etc. In the latter the pupils will probably be normal.

If *lumbar puncture* reveals a lymphocytosis and a distinctly albuminous cerebro-spinal fluid, it points to a subacute or chronic inflammation, and therefore suggests general paralysis, tabes, or cerebral syphilis. On the other hand, an excess of polynuclear cells would suggest an acute inflammation ; while under normal conditions very few cells would be found, and the fluid would contain little or no albumen.

Prognosis.—Temporary improvement, and even apparent recovery, may occur, but in the main the disease is progressive, and death generally ensues in a few years.

Treatment.—Confinement in an asylum is often essential. In any case, the patient should at once be relieved of any mental labour, anxiety, etc., connected with business. If he can be managed by his friends, he should be kept in a quiet country place and carefully attended to. If the syphilitic infection is recent, specific treatment should have a fair trial. For sleeplessness, restlessness and convulsions, cold applications to the head, and bromides are indicated.

iv. Disorders of Speech.

Disturbance of speech may result from disease of either the higher or the lower neurons of the path to the muscles concerned in articulation. These muscles are for the most

part innervated from both hemispheres, so that permanent paralysis is only produced by bilateral disease. Disease in the medulla and lower pons is often bilateral, and gives rise to a defect in articulation (one symptom of *bulbar paralysis*). The patient uses the correct words, and arranges them in the correct order, but he cannot pronounce them properly. Thus paralysis of the lips will impair or prevent the pronunciation of the letters *p* and *b ;* palsy of the tongue will interfere with the pronunciation of *l* and *t*, and paralysis of the palate will cause a nasal tone of voice. In slighter cases of palsy involving these parts, the final syllables or consonants of words are apt to be omitted (*elisive* speech), or run into the following word (*confluent* articulation). Sometimes, on the other hand, words or syllables are separated too much (*staccato* or *scanning* articulation).

Defects of articulation, such as have been described, constitute DYSARTHRIA or ANARTHRIA. The same result is occasionally produced by disease of both cerebral hemispheres involving the upper neurons of both sides in any part of their course (*pseudo-bulbar paralysis*). One-sided disease of the brain may cause a similar temporary loss.

APHASIA, on the other hand, is a disorder of speech resulting from disease of the cerebral hemisphere, and characterised by errors in the number and arrangement of words. Their form or pronunciation may also be defective, but this is not essential. Aphasia is due to a lesion which destroys or isolates, wholly or in part, one or more of the four cortical speech centres (Fig. 39, p. 732). In right-handed people these centres are situated in the left hemisphere, the right hemisphere not being educated to intellectual speech. If speech is lost through destruction of these centres, it can only be regained by education of the opposite hemisphere, an occurrence which takes place readily in childhood, but very imperfectly as a rule in adult life. In left-handed individuals, the right hemisphere is the one which is ordinarily the seat of intellectual speech processes. Nevertheless the right hemisphere in right-handed individuals, and probably the left hemisphere in left-handed persons, seem to take some small share in speech processes, but this is in an automatic manner, and probably only in association with, or under the

instigation and guidance of, the opposite hemisphere. When the hemisphere which has been educated for speech is thrown out of action by disease, evidences of a feeble training in speech processes may be manifested by the opposite side of the brain.

Motor aphasia is loss of the power of giving expression to intellectual language either by speaking (*aphemia*) or by writing (*agraphia*). *Sensory aphasia* is loss of the power of understanding language, either on hearing it (*word-deafness*) or by reading it (*word-blindness, alexia*). A person who is word-deaf speaks in a disordered way (*auditory aphasia*), and a person who is word-blind is also apt to show disorder of speech (*visual aphasia*).

In addition to the two kinds of motor, and the two kinds of sensory aphasia, there is a fifth variety which has been called *conduction aphasia*, *amnesic aphasia*, or *loss of memory for words*. In this condition the motor speech processes are not lost, and there is no sensory aphasia, but the patient has a difficulty in recalling words (*verbal amnesia*), and often brings out wrong words (*paraphasia*). It has been attributed to interruption of the fibre tract connecting the sensory and motor speech centres by disease of the island of Reil ; but a much more satisfactory theory is that there is a defect of function in the auditory speech centre.

In *total aphasia*, speech is completely lost in consequence of destruction of all the speech centres.

In connection with aphasia, mention may be made of *mind-blindness*, a condition in which the patient is not merely word-blind, but object-blind. He is unable to recognise the nature of objects which he sees, though he may identify them by his other senses. Similarly the expression *mind-deafness* has been introduced for a condition in which the patient is not merely word-deaf, but is unable to recognise the nature and significance of other sounds which he hears.

The centre by which heard language is understood (*auditory speech centre*) is in the posterior half of the first temporal or temporo-sphenoidal convolution, close to the cortical auditory centre. This is the most important of all the speech centres. It is naturally the earliest to become active in the child, for it is here that the memories of words

heard are stored up. In ordinary circumstances, when a healthy person speaks, the words he employs appear to be revived in this centre, which thereupon transmits the necessary impulses to the centre for giving utterance to the words.

The centre by which language is spoken (*motor speech centre*, Bastian's *glosso-kinæsthetic centre*) is Broca's convolution, the posterior part of the third left frontal gyrus. Fibre tracts pass from the auditory to the motor speech centre by way of the white substance of the island of Reil. Moreover, commissural fibres pass through the corpus callosum from Broca's convolution on the left side to the corresponding part of the cortex on the right side, so that when the path from Broca's convolution to the bulbar nuclei is interrupted by disease, motor speech processes can be effected by Broca's centre acting on the cortex and motor path of the opposite hemisphere. The motor speech centre does not coincide exactly with the cortical centre for the tongue and lips. The former (Broca's convolution) is in the posterior part of the lower frontal gyrus, and possibly also in the neighbouring part of the island of Reil. By means of short association fibres, it transmits impulses for spoken language to the motor centres for articulation in the lower part of the ascending frontal convolution.

The centre by which printed and written words are understood (*visual word centre*) is in the angular region, where the highest visual centre is also placed. The memories of words seen are stored up here.

Until recently there was some doubt as to the existence of a *graphic centre* by which words are written (Bastian's *cheiro-kinæsthetic centre*), although this was recognised by some authorities, and was supposed to correspond to the cortical centre for movements of the hand. The older view was that there was no graphic speech centre, and that in writing the ordinary centre for the hand operated in obedience to impulses emanating from the motor speech centre in Broca's convolution. Clinical and anatomical evidence, however, has now made it clear that there is a graphic centre independent of Broca's centre, viz., at the base of the second left frontal convolution in right-handed persons. De-

struction of this centre causes pure agraphia without any other form of aphasia, and without paralysis of the arm.[1] On Bastian's view, the cheiro-kinæsthetic centre is more intimately connected with the visual word centre than with any other.

It must be borne in mind that the various centres are closely connected with one another by tracts of nerve fibres, and that disorders of speech may be due to interruption of connecting paths as well as to lesions of the centres themselves.

The lesion which causes aphasia usually involves more than one of the speech centres or fibre tracts, so that in practice it is frequently impossible to diagnose its exact situation and extent. Another difficulty is introduced by the imperfect speech that remains, since this may be due to partial escape of the speech centres, or to activity of the opposite hemisphere, or to both causes.

In investigating a case of aphasia, it is of course necessary to make sure that inability to understand spoken or written language is not due to deafness or blindness.

In *word-deafness*, from disease of the first temporal convolution, the patient does not understand heard words, cannot repeat words, and cannot write to dictation. Voluntary speech especially is lost (*aphasia*) or disordered (*paraphasia*), because voluntary speech is chiefly initiated in the auditory centre. The patient does not recognise his own mistakes in speaking. Writing is also lost (*agraphia*) or disordered (*paragraphia*). The patient is often unable to read (*alexia*), but this is probably due to simultaneous disease of the visual centre.

If a lesion is so situated as to cut off the auditory speech centre from both cortical centres for hearing, without damaging the speech centre itself, the result is *subcortical word-deafness* (sometimes called *subcortical sensory aphasia* or *pure word-deafness*). The patient is unable to understand what he hears, or to repeat words, or to write to dic-

[1] See Gordinier, *Amer. Journ. Med. Sc.*, September, 1903, p. 490. Destruction of the cortex of Broca's convolution may cause aphemia without agraphia.

tation ; but since his auditory speech memories remain stored in the uninjured centre, he can speak and write on his own initiative. He is also able to read.

In *motor aphasia*, or *aphemia*, from destruction of Broca's centre, there is partial or complete inability to speak spontaneously or to repeat words. The patient understands what is said to him, and recognises his own mistakes in speech. The power of writing, even if the hand is not paralysed, is often lost (*agraphia*), but this is not always the case. The intellectual processes are not necessarily impaired to any great extent.

In *word-blindness*, from a lesion in the angular region, there is inability to understand written or printed words. Speech may be good, and spoken language is understood. In ordinary cases, the patient is unable to write, except perhaps his own name, and he cannot copy properly. In other cases, however, he can write, and when this is so, he can write as well with eyes closed as with eyes open, and very shortly after he has written, he is unable to read the writing. Such a patient is spoken of as a ' strong auditive ' and a ' weak visual.' He may have been accustomed to write much, and the auditory centre, in which the words originate, has learned to incite the activity of the graphic centre directly, with very little assistance from the visual centre. In the average individual the writing centre is most likely to be incited to action by the visual centre, and the motor speech centre by the auditory centre.

If a lesion is so situated as to cut off the visual word centre from the subordinate cortical centres for vision, *subcortical alexia* (*pure word-blindness*) results. The patient cannot read, but he can speak, and if he is a ' strong auditive,' he can write. Hemianopia is likely to be present along with this variety of aphasia.

Agraphia as an isolated speech defect is of rare occurrence. It was produced in Gordinier's case (see footnote, p. 750) by a tumour of the posterior end of the second left frontal convolution.

In aphasia the loss of speech is rarely complete, and, in any case, it is voluntary speech which suffers most. In

sensory aphasia especially, the patient may use a considerable number of words in a more or less automatic way. These automatic utterances are doubtless due in great part to the activity of the uninjured hemisphere. The patient may from the first, or after a time, be able to express himself by gesture. Simple words such as 'yes' and 'no' are often retained, and it is to be noted that these may be used wrongly. The automatic speech which results from the activity of the right hemisphere sometimes takes the form of *recurring utterances*. These may be the last words uttered by the patient before speech was lost, or possibly the words he was about to utter. In many cases the right hemisphere becomes educated for intellectual speech, and occasionally disease of this side of the brain has again caused aphasia.

Etiology.—The various speech centres are supplied by the middle cerebral artery, and persistent aphasia is generally due to acute softening which results from obstruction of one or more branches, or of the main trunk of that vessel. The cause of obstruction may be embolism or thrombosis. The first branch supplies the inferior frontal convolution with the motor speech centre. The second supplies most of the ascending frontal convolution, and therefore part of the writing centre. The third supplies the ascending parietal convolution and the superior parietal lobule. The fourth supplies the supramarginal and angular gyri, the posterior end of the superior parietal lobule, and the posterior portions of the first two temporal convolutions ; these regions include the visual and auditory speech centres. The middle cerebral also gives branches to the anterior portions of the first two temporal gyri. The posterior part of the angular gyrus seems often to get part of its blood-supply from the posterior cerebral artery, and accordingly this region is seldom completely destroyed by obstruction of the middle cerebral alone.

Hæmorrhage involving the cortex is an occasional cause of persistent aphasia, and hæmorrhage in the region of the internal capsule is a common cause of transient aphasia. Tumour of the brain and tubercular meningitis may also

cause aphasia, and occasionally the symptom results from functional causes — *e.g.*, strong emotion in childhood. Aphasia may be an element in an attack of migraine. It may also apparently be induced by numerous agencies, such as morbid blood-states and overwork, which lower the functional capacity of the speech centres, either by poisoning them or by exhausting them.

Aphasia is often associated with other results of vascular disease within the head, such as hemiplegia, hemianopia, etc.

Prognosis.—In children this is favourable, since the opposite hemisphere readily becomes educated for intellectual speech. In adults the prognosis is not so good, but the ultimate condition varies greatly in different individuals. On the whole, recovery appears to take place more readily in sensory than in motor aphasia. But the loss, whether it amounts to word deafness or to total aphasia, may persist unchanged.

Aphasia is not a bar to will-making, if it is clear that the patient retains sufficient mental capacity to appreciate all the details of the procedure. In February, 1900, the Probate Court recognised as valid a will made by a lady who had complete motor aphasia with agraphia and word deafness, though the visual speech centre was intact. Her solicitor wrote out two sets of cards, one bearing the names of items of property, the other bearing the names of her relatives. The lawyer put down one of the property-cards, and the lady, after looking through the relative-cards, selected and put down the card with the name of the friend to whom she desired to allot that particular portion of her estate. This process was repeated till all the property was disposed of.[1]

Treatment.—This consists firstly in treatment of the cause of the aphasia, and secondly in endeavouring, after a period of rest, to re-educate the patient in speech, beginning with the simplest objects and words.

[1] Aphasia and will-making appear in fiction in Dumas's work, 'The Count of Monte Cristo,' chap. lix.

v. Hemiplegia.

Hemiplegia, or paralysis of one half of the body, is the result of a lesion which damages or compresses the motor path at any part of its course within the cranium, from the cortex to the decussation of the pyramids. In a well-marked case of hemiplegia such as results from a hæmorrhage in the region of the internal capsule, the leg, arm and lower part of the face are completely paralysed on the side opposite to the lesion, and the tongue when protruded deviates towards the weak side through the action of the healthy genioglossus muscle. Some muscles of the trunk, and perhaps those in the upper part of the face, may be weakened. Thus the patient may be unable to close the eye on the affected side as firmly as the other eye, and there may be slight ptosis. Other muscles—*e.g.*, those of ordinary respiration and of mastication—seem unaffected. If the patient survives, the weakened muscles regain their strength, some of the paralysed muscles recover part of their power, and others (in many cases) remain permanently paralysed.

These facts are explained by *Broadbent's law* in the following way. Muscles which are habitually innervated from both hemispheres continue to act in hemiplegia. Those which are connected anatomically with both hemispheres, though habitually stimulated only by one hemisphere, lose their power only temporarily. Those which are connected anatomically with only one hemisphere remain paralysed when the motor path is severed. It will be remembered that some fibres of the pyramidal tract pass at the pyramids into the crossed pyramidal tract of their own side instead of crossing to the opposite side. This arrangement allows of upper neurons from each hemisphere coming into relation with lower neurons in both halves of the spinal cord. In other words, motor cells in each anterior horn of the cord are related to upper neurons, whose cells are in both hemispheres. Broadbent's original view, which is not so probable as the explanation just given, was that commissures connected the upper ends of the lower neurons, and that the upper neurons transmitted impulses not only to the

corresponding lower neurons, but also, if need be, along the commissures to the lower neurons of the opposite side.

The greater the degree to which muscles on the two sides of the body are accustomed to act together, the more intimately are they bilaterally connected with the brain. This applies to such muscles as those of mastication and respiration, and to a considerable extent to the muscles of the lower limbs. On the other hand, muscles which are accustomed to act on one side of the body at a time are connected almost solely with the opposite cerebral hemisphere ; and the more highly differentiated a movement is, as in the case of many delicate movements of the hand, the more completely is its connection restricted to the opposite side of the brain. It is natural therefore that highly differentiated, carefully trained and unilateral movements should be completely lost as a result of lesions of the upper neurons in the opposite side of the brain ; while coarse, more or less automatic and bilateral movements are at most weakened by a unilateral cerebral lesion.

The arm generally suffers much more than the leg from permanent weakness.

The weakness of the lower part of the face is not so conspicuous in emotional movement (*e.g.*, smiling) as in voluntary movement (*e.g.*, showing the teeth).[1] This is an important point of distinction between hemiplegic weakness and that due to disease of the facial nerve, since in the latter case the two kinds of movement suffer alike. What has been said of emotional movement applies also to associated movement as seen in those whose mouth is drawn to the side when the hand grasps an article firmly.

A frequent symptom in the early stage of hemiplegia is loss of conjugate movement of the head and eyes to the opposite side from the lesion, so that they remain turned towards the side of lesion, and the patient cannot look towards his paralysed limbs. This movement, however, is

[1] The converse of this, viz., loss of emotional with preservation of voluntary movements of the face, has been noted as a symptom of disease (*e.g.*, tumour) in the optic thalamus. It also occurs in infantile hemiplegia as a result of a lesion of the cortical motor area.

represented on both sides of the brain, and the paralytic deviation passes off before long.

The cutaneous reflexes are often diminished or lost on the paralysed side. This loss is present from the outset.

Special features distinguish hemiplegias due to lesions in particular situations. Thus the form already described as typical, with palsy of the leg, arm and lower part of the face on one side, results from a *lesion above the middle of the pons*, since the upper neurons destined for the nucleus of the seventh nerve cross the middle line at that level. A lesion in the *lower part of the pons* would miss these, but might damage the lower neurons (in the facial nerve or its nucleus) for the face muscles on the side of the lesion—*i.e.*, on the side opposite to the palsied limbs. This constitutes one form of *crossed paralysis* or *alternate hemiplegia*.

Disease in the *medulla* paralyses the limbs on the opposite side, and probably the tongue on the same side as the lesion, owing to damage to the hypoglossal root-fibres. In this case the tongue deviates towards the side of lesion, or away from the paralysed limbs—another instance of *crossed paralysis*. Both sides of the face escape, except perhaps the lips, whose nerve-supply comes from the hypoglossal nucleus through the facial nerve.

Disease in the *upper half of the pons* involves the lower part of the face, the arm and the leg on the opposite side ; and frequently the fifth nerve on the same side as the lesion —another instance of *crossed paralysis*.

Disease in the *crus cerebri* involves the limbs and lower face of the opposite side, and the third nerve on the side of lesion—again a *crossed paralysis*.

In distinguishing between lesions near the internal capsule and lesions near the cortex, two circumstances are available to guide us. (1) In the *internal capsule*, the various fibre tracts of the motor path (upper neurons for face, arm and leg muscles) are crowded together into a small space, so that a lesion damaging one is apt to damage all ; and, moreover, such a lesion, being often relatively small, may be associated with comparatively slight general symptoms. On the other hand, at the *cortex*, the upper ends of the neurons are spread over a wide expanse of

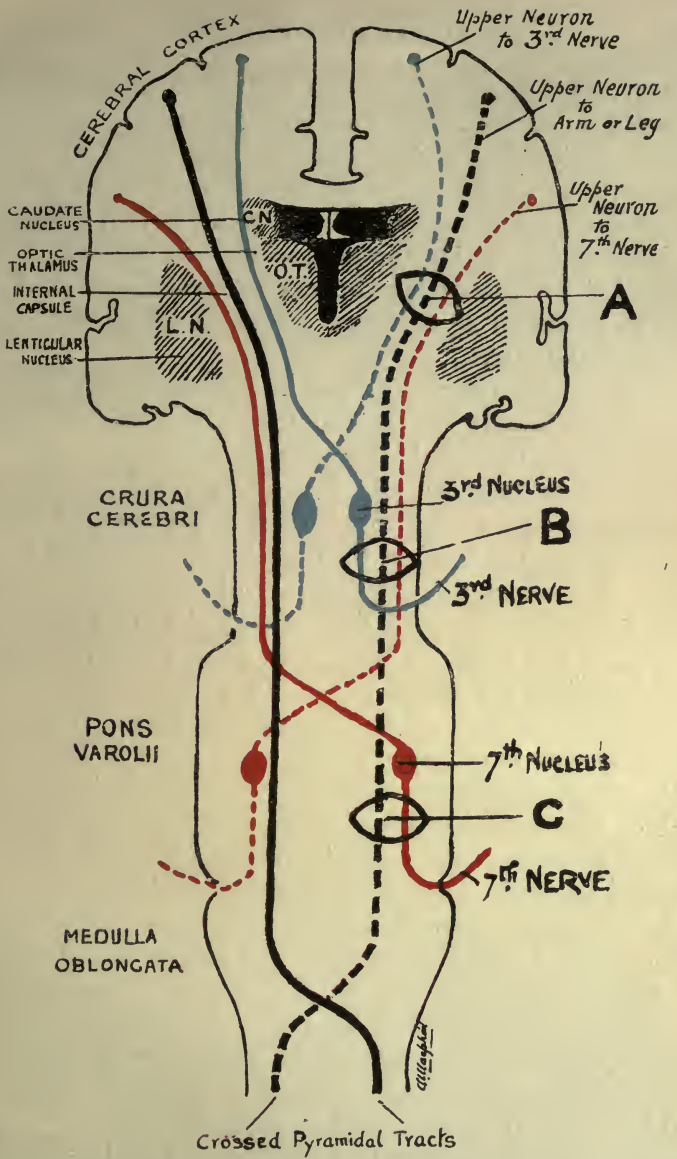

FIG. 42.—DIAGRAM OF THE MOTOR PATH FROM THE CEREBRAL CORTEX TO THE MEDULLA.

The path to the ocular muscles is indicated by blue, to the facial muscles by red, and to the limbs by black. The paths originating in the one hemisphere are indicated by continuous lines, while those starting from the opposite hemisphere are represented by interrupted lines.

It will be seen that a lesion at A (*internal capsule*) involves upper neurons only, so that the paralysis which it causes is on the opposite side (limbs and lower face).

A lesion at B (*crus*) involves the upper neurons for the limbs and face, which are thus paralysed on the opposite side; but it also involves the lower neurons for the ocular muscles, and in this way causes oculomotor paralysis on the side of lesion.

A lesion at C (*pons*) involves the upper neurons for the limbs, which are thus paralysed on the opposite side; but it also damages the lower neurons for the face, which is therefore paralysed on the side of lesion.

cortex, and it is easy for a lesion to damage one set (causing *monoplegia*) without interfering with the others ; and, moreover, a lesion which does interfere with them all, being extensive, is likely to be associated with grave general symptoms. (2) The second circumstance is that lesions of, or immediately under the cortex are specially apt to be associated with recurring convulsions, which are attributable to irritation of the grey matter in which the upper neurons take origin.

So far as the *internal capsule* is concerned, the part involved can usually be inferred from the extent to which motion, sensation and the special senses are affected, separately or together (see Fig. 41, p. 735). It will be remembered that the neurons related to the face and tongue are at the genu or bend of the capsule ; those related to the upper limb are in the anterior part of the posterior limb ; those related to the lower limb occupy the middle third or thereby of the posterior limb ; and those related to sensation, including the special senses, are in the posterior third of the posterior limb, at the sensory crossway. If the sensory crossway is completely destroyed, there is anæsthesia with loss of the special senses on the opposite side of the body. The visual loss takes the form of lateral or homonymous hemianopia ; the patient, when his face and eyes are directed forwards, cannot see his paralysed limbs.

A lesion of the *cortex* may involve only a part of the motor area. Thus a syphilitic gumma may compress and irritate the centre for movements of the arm, and thus cause localised convulsions and paresis of the limb. Paralysis of a single limb is spoken of as a *monoplegia*, and is usually due to a cortical lesion, since at a lower level the tracts for the limbs and face converge and are apt to suffer together.

It occasionally happens that no lesion is found after death to account for hemiplegia. In other exceptional instances, a lesion is found in the brain on the same side as the paralysis (*collateral paralysis*). Morgagni suggested that cases of the latter kind might be due to absence of the decussation of the pyramids.

Muscular rigidity is frequently recognised in association with hemiplegia. Gowers recognises four varieties, of which

the second and third were originally pointed out by Todd. (1) Initial rigidity is due to the irritation caused by the lesion. It comes on at once, and lasts for a few hours, but is often absent. (2) Early rigidity comes on after a few days, and lasts for a few weeks. It is due to irritation of the motor neurons by the inflammatory process set up by the lesion. The posture of the limbs is that of rest. (3) Late rigidity is accompanied by exaggeration of the tendon-jerks, Babinski's sign, ankle clonus, etc. It is characterised by adduction of the upper arm, flexion of the elbow, flexion and pronation of the wrist, flexion of the fingers and extension of the knee. It comes on within a few weeks, and lasts for months or years. If the power of the limbs is not restored, this rigidity becomes permanent; and since, in the case of the leg, it is extensor, it is much more favourable to walking than the mere loss of power would be. (4) Structural rigidity is the result of the tissue changes which take place in muscles which have long been fixed by late rigidity.

The paralysed limbs are sometimes colder or warmer than the others. They may also be red or livid, slightly swollen, and the seat of increased perspiration. It is ominous when there is a marked tendency to trophic changes; these naturally occur most readily where the skin is compressed by the weight of the body, as over the malleolus, trochanter, etc. An important bedsore is one which develops in the gluteal region of the paralysed side in certain cases of hemiplegia (*acute decubitus*). When hemiplegia occurs in childhood, the paralysed limbs do not attain full development.

Since the lesion involves only the upper neurons, so far as the limbs are concerned, wasting and electrical changes are not to be expected in the paralysed muscles. In a number of cases, however, a certain amount of wasting of the muscles, and a certain degree of change in their electrical irritability—either in the way of increase, or in the way of diminution—do take place. Gowers explains this on the view that the secondary descending degeneration of the pryamidal tract (upper neurons) is irritative in character, and influences to some extent the nutrition of the cells of the lower neurons.

In cases where some voluntary power is regained, the arm,

and to a much less extent the leg, may become the seat of disordered movement. The most common variety is that known as *mobile spasm* or *athetoid movement*.[1] In this condition the muscles are the seat of tonic spasm which slowly changes in degree in the different muscles, so that the digits in particular show constant, slow, irregular movements. The upper arms tend to be adducted, and sometimes the hand is drawn behind the back.

In some instances the movements are more jerky, and the arm and leg may suffer as much as the fingers and toes. Such cases have been called *post-hemiplegic chorea*.

These disorders of movement after hemiplegia are far more common when the disease sets in in childhood than when it occurs in adult life. They are also far more common after arterial obstruction and cerebral softening than after arterial rupture and cerebral hæmorrhage.

Etiology.—The causes of hemiplegia are various. As Gowers points out, the mode of onset is most important in diagnosis. A sudden onset means a vascular lesion, viz., obstruction or rupture of a vessel ; and obstruction may be due to either embolism or thrombosis. An acute onset means inflammation, either of the brain or of the meninges. A gradual onset means tumour or sclerosis. Thrombosis in connection with disease of the cerebral arteries may cause hemiplegia of gradual onset, but careful observation or inquiry will sometimes show that the gradual loss of power is not a uniform one, but consists of a succession of sudden steps.

Prognosis.—If hemiplegia is due simply to compression of neurons by a hæmorrhagic effusion, recovery may be complete. Hemiplegia may also be recovered from if it is due to softening in the neighbourhood of the neurons, as in syphilitic vascular disease. Destruction of the motor neurons excludes recovery except of muscles which are connected with both sides of the brain. Hemianæsthesia in childhood, even though due to destruction of sensory neurons in the brain, is recovered from if the child lives,

[1] The term *athetosis* is sometimes used in the same sense, but was originally used of similar movements setting in without preceding hemiplegia.

doubtless through the development of other sensory paths. In many of the cases so commonly met with in adult life, the arm is permanently paralysed, but the patient is able to walk, and he retains good health until he is seized with a second or later attack, or is cut off by chronic kidney disease or some intercurrent illness.

Treatment.—The treatment is at first simply that of the cause of the paralysis. After some weeks, however, when the cerebral lesion may be regarded as healed, nervine tonics such as strychnine may be given internally, and the affected limbs, if voluntary power has not returned, should be subjected to massage and electrical stimulation. These measures, however, must be begun with caution, so as not to fatigue or excite the patient.

vi. Intracranial Hæmorrhage.

Hæmorrhage may take place into the substance of the brain, into the ventricles, or into the membranes. All three forms are commonly included under the designation 'cerebral hæmorrhage.'

Intracerebral Hæmorrhage.

Etiology.—Cerebral hæmorrhage in the strict sense is occasionally traumatic, but as seen by the physician it is generally spontaneous. Among the causes, age is the most important, the vast majority of cases occurring after forty. Heredity, gout, lead poisoning, chronic alcoholism, overeating and excessive effort are all important because they are causes of high arterial tension, hypertrophy of the heart, and arterial degeneration, either directly or by inducing kidney disease. Bright's disease, by causing arterial degeneration and cardiac hypertrophy, favours the occurrence of cerebral hæmorrhage at an earlier age than forty. Males suffer from cerebral hæmorrhage more frequently than females. Embolism and syphilis are causes of hæmorrhage as being causes of aneurysm of the larger arteries, which may develop in early life as well as at a later period. Cerebral hæmorrhage may also occur in connection with such diseases as leucocythæmia, purpura, scurvy and hæmophilia. Sometimes the rupture is immediately due to mental or

physical excitement. Hæmorrhage sometimes occurs into the substance of tumours of the brain.

As the immediate cause of rupture of a cerebral artery, one or both of two agencies will almost always be recognisable, viz., weakening of the vessel wall, and increased distending force of the blood within the vessel.

Morbid Anatomy. — Intracerebral hæmorrhage is most frequent in or near the corpus striatum, then in the centrum ovale, and then in the cortex. One of the lenticulostriate branches of the middle cerebral is particularly prone to rupture (see p. 737). The blood forms a cavity for itself by tearing the brain tissue. This cavity varies in size, and has rough walls of lacerated brain substance. The blood is clotted, and portions of brain tissue may be found in its midst. The hæmorrhagic effusion compresses nerve fibres which it does not actually destroy. It causes bulging of the fornix to the opposite side, and it causes the convolutions on its own side to be flattened and anæmic. It may tear its way into the lateral ventricle, and then pass into the opposite ventricle and the third ventricle, and thus reach the Sylvian aqueduct and fourth ventricle.

Many cases in which the hæmorrhage does not burst into the ventricles survive, and changes then take place in the blood-clot and the surrounding tissues. The clot changes in colour from reddish-black to brown and to reddish-yellow. The surrounding tissue undergoes inflammation, and produces a fibrous tissue wall for the cavity. The latter has a smooth lining, and often persists as a cyst containing fluid. Hæmatoidin crystals may long be recognisable in and around the seat of lesion. A cyst with a firm wall usually takes several months to develop.

The ordinary form of spontaneous cerebral hæmorrhage is always due to the rupture of one or more *miliary aneurysms*. By washing away the brain substance, aneurysms of this kind become just visible to the unaided eye. They develop on the minute arteries in consequence of atrophy of the middle coat. In other forms of intracerebral hæmorrhage and in meningeal hæmorrhage the appearances will vary according to the site and cause of the lesion.

A hæmorrhage which severs nerve fibres is of course followed by secondary degeneration. Thus, when the posterior limb of the internal capsule is torn, degeneration takes place after a time below the lesion, and can be traced downwards through the crus, pons and pyramid of the medulla into the lateral pyramidal tract of the opposite side. It can also be traced into the direct pyramidal tract and, to a slight extent, into the lateral pyramidal tract, on the side of the lesion.

Symptoms.—In rare cases there are premonitory symptoms such as tingling in the limbs or headache, attributable to a minute hæmorrhage or to coincident atheroma. The symptoms of any considerable hæmorrhage are of two kinds, the one being general and transient, the other local and more lasting.

(1) The most important general symptom is sudden loss of consciousness. Occasionally the patient loses consciousness at once and falls, but more often he feels giddy or has headache and becomes weak in one side; or his speech becomes indistinct and he then becomes insensible. There is sometimes a convulsion at the outset. This unconsciousness and its associated phenomena constitute *apoplexy*.[1] The condition varies greatly in different cases. In a few, consciousness is not actually lost; in others the unconsciousness or coma is profound, reflex action is lost and the evacuations are passed involuntarily; whilst in many the condition is intermediate between these two extremes. Respiration is laboured, the pulse slow, and the skin wet with sweat. The temperature falls below the normal. In well-marked cases the flaccid cheeks are puffed out with each expiration, and the paralysis of the palate renders the breathing snoring or stertorous.

Occasionally the effusion takes place more slowly, and consciousness is lost gradually in the course of hours (*ingravescent apoplexy*).

[1] In old people attacks may occur which resemble the apoplexy of cerebral hæmorrhage, but are not associated with localising symptoms, or with any recognisable lesion in the brain. These are sometimes known as *simple apoplexy* or *serous apoplexy*.

(2) The most important local symptom is hemiplegia involving the limbs on the side opposite to the lesion. Even when the patient is unconscious, the limbs can generally be recognised as more flaccid than those of the healthy side. The cutaneous reflexes are often diminished or lost on the paralysed side. There may also be paralytic deviation of the head and eyes to the side of the cerebral lesion.

In many cases, signs of returning consciousness will be observed in the course of a few hours. The temperature rises again to the normal or above it, and thereafter for a time there may be some headache and delirium. In cases, however, where the effusion continues to increase, coma deepens till death occurs, and the temperature may remain at a low figure. Death is generally due to interference with the respiratory centre, and the accumulation of moisture in the air-passages.

If the patient survive for two or three days, inflammatory changes take place around the lesion, and the temperature rises. The pulse is accelerated, delirium may supervene, and early rigidity is present. Serious trophic lesions of the skin may take place at this stage—*e.g.*, an acute bedsore (*acute decubitus*) on the buttock of the paralysed side. The patient may die in this stage. But often these phenomena are less marked, and the patient recovers from all except the local symptoms. Moreover, these too usually diminish with the lapse of time, partly because the effusion compresses for a time nerve fibres which it does not tear and which afterwards regain their conducting power, and partly because many movements become innervated from the opposite hemisphere. Thus hemianopia and conjugate deviation of the head and eyes may be present at first, and afterwards pass off.

Hemiplegia, then, is the usual localising manifestation of cerebral hæmorrhage, and the situation of the lesion can usually be recognised by the special features present, as explained in the section on hemiplegia. If the posterior part of the posterior limb of the internal capsule is involved, sensation will be affected, and perhaps the special senses. In very slight cases, the paralysis may disappear within

days or weeks, but as a rule some hemiplegic weakness remains permanently. After a couple of weeks, the knee-jerks, and especially that on the paralysed side, become permanently exaggerated, owing to secondary degeneration of the pyramidal tract. The leg, being innervated from both hemispheres, usually recovers more than the arm; and as the late rigidity in the leg is extensor, the power of walking is often regained. The gait, however, is characteristic, the limb being swung round and the foot dragged. In many cases the hand remains permanently powerless, and fixed by the late rigidity.

Ventricular hæmorrhage gives rise to profound apoplexy. *Meningeal* hæmorrhage is sometimes associated with repeated convulsions which may be chiefly or only on one side.

Diagnosis.—There may be great difficulty in distinguishing between apoplexy, alcoholic intoxication, uræmic coma and opium poisoning; all the more since intoxicated persons may be seized with spontaneous cerebral hæmorrhage or have their skulls fractured through accident or malice, and also because individuals who are beginning to feel unwell are apt to take or to get some kind of alcoholic drink. The odour of the breath or of vomited matter is therefore often fallacious. If the history of the case can be obtained, this may be sufficient. Unilateral flaccidity or rigidity, and conjugate deviation of the head and eyes are important evidence of a cerebral lesion. The head should be carefully examined for any signs of injury, and the urine should be examined for albumen and sugar. The condition of the heart and arteries may be very significant.

Alcoholic coma is usually less intense than that of apoplexy, and sets in gradually. If the patient struggles much, alcohol is likely. Convulsions at the outset of the attack and localising symptoms indicate something more than this intoxication.

Uræmic coma is often of very gradual onset, and is thus for a considerable time less deep than that of apoplexy The temperature tends to become lower and lower, unless convulsions or inflammatory complications are present.

But in acute nephritis especially, convulsions may set in abruptly and be associated with profound coma. Localising symptoms do not absolutely exclude uræmia. The history of the case, and a careful examination of the heart, arteries, urine and retinæ may give valuable information, but it must be remembered that cerebral hæmorrhage is common in the subjects of chronic renal disease.

Inequality of the pupils is suggestive of a cerebral lesion, but otherwise the condition of the pupils is not of great value in the diagnosis. The pupils may be strongly contracted in opium poisoning, and also in hæmorrhage into the pons ; but they may be normal or dilated in alcoholic, opium and uræmic poisoning, and in apoplexy.

The diagnosis between arterial rupture and arterial obstruction is important, and sometimes very difficult ; but this will be considered after an account has been given of the latter condition.

Prognosis.—If the coma does not lessen within twenty-four hours, recovery is unlikely. Cheyne-Stokes breathing, œdema of the lungs, and an early and considerable rise of temperature are unfavourable signs. In the period of reaction, considerable fever and delirium, return of the drowsiness, and early sloughing of the skin are unfavourable symptoms. As to the ultimate recovery of power, it may be necessary to wait for a month or two before an accurate forecast can be made.

In the event of recovery there is always the risk of a second attack of the same kind ; or death may result from kidney disease or some intercurrent affection.

Treatment.—The patient should lie with the head and shoulders raised. A purge of calomel or croton oil and a diuretic should be given. An ice-bag may be applied to the head, and a hot bottle to the feet. Professional opinion is undecided as to the value of venesection, but if the heart were acting strongly, the pulse incompressible and the coma deep, a trial of this method would be justifiable. Compression of the carotid has also been recommended. During the period of inflammatory reaction, careful nursing is of great importance, and every precaution must be taken

to avert bedsores. The bladder must be attended to. If there is much fever, ice may be applied to the head. Headache is to be treated by the local application of cold, and by phenacetin or citrate of caffeine given internally. The diet must be light. The patient should rest completely for about six weeks, namely, till the cerebral lesion has had time to heal; after which, stimulating measures, such as massage and faradism, may be applied to the paralysed limbs, and strychnine may be given internally.

The patient must for the future lead a quiet life, observing strict moderation in food, drink and exercise, and avoiding bodily and mental excitement.

Hæmorrhage into the Pons Varolii.

Convulsions are very common at the outset and usually affect all the limbs, but may be confined to the two lower limbs. There may be tonic as well as clonic spasm. The palsy, like the spasm, is usually bilateral, and anæsthesia is often present in addition. Loss of consciousness may or may not be present at the commencement. The pupils are often strongly contracted from irritation of the nuclei of the third nerves, but sometimes they are dilated owing to paralysis of these nuclei. Vomiting is common. The temperature may begin to rise within an hour, and continue to rise quickly until a great height is reached.

Occasionally a small hæmorrhage causes unilateral symptoms, viz., hemiplegia involving the limbs on the side opposite the lesion, and one or more of the cranial nerves (*e.g.*, fifth, sixth, or seventh) on the side of lesion.

Death generally takes place more quickly than in ordinary cerebral hæmorrhage, but if the lesion is small, life may be spared.

Hæmorrhage into the Medulla Oblongata.

This causes death very quickly, and even, it is said, instantaneously. If the patient survives the onset, which is quite exceptional, he presents symptoms like those of bulbar paralysis.

Hæmorrhage into the Cerebellum.

This, like bulbar hæmorrhage, is a rare occurrence. The mode of onset may suggest ordinary cerebral hæmorrhage. The hemiplegia may be on the opposite side or on the same side, according as the pressure is exerted upon the pons or upon the medulla. The nerves arising from these parts may be involved. In some cases, however, there is no hemiplegia. Vomiting is very common.

Death may result from the hæmorrhage bursting into the fourth ventricle, but in other cases the patient recovers. He then becomes free from the symptoms of pressure on the pyramidal tract and cranial nerves, though he may continue to suffer from disturbance of equilibrium.

Ventricular Hæmorrhage.

Ventricular hæmorrhage is seldom primary. It is usually secondary to intracerebral hæmorrhage. In most cases, the hæmorrhage ruptures into the lateral ventricle, from which it passes into the opposite lateral ventricle and into the third and fourth ventricles. Occasionally a hæmorrhage bursts directly into the fourth ventricle—*e.g.*, from the cerebellum.

The symptoms are those of a severe type of apoplexy. In secondary cases, the original apoplectic symptoms may have begun to subside, when they suddenly reappear with renewed or increased intensity. If they have not subsided, they may become more intense. There may be a renewed fall of temperature and slowing of the pulse for a short time. There may be rigidity of the limbs on one or both sides.

When ventricular hæmorrhage is primary, the symptoms are at first, in most cases, suggestive of intracerebral hæmorrhage. There may be hemiplegia for a time, and then when the effusion reaches the opposite ventricle, paralysis involves both sides of the body.

Recovery takes place only in exceptional cases of the primary form.

Meningeal Hæmorrhage.

Etiology.—Meningeal hæmorrhage may be extradural, subdural, or subarachnoid. It is almost as common before as after forty years of age. It is about three times as common in males as in females. It occurs under the same conditions as to age, high tension, etc., as intracerebral hæmorrhage (see p. 761). It may also be due to injury to the head; to rupture of an aneurysm of a large artery; or to some hæmorrhagic disease. It sometimes results from compression of the skull at birth (see below, Cerebral Diplegia), and it is seen in the hæmatoma of the dura mater (p. 738). The extradural form is almost always traumatic.

Symptoms.—If the hæmorrhage is the result of injury, the symptoms of the injury may mask those of the hæmorrhage, particularly if the latter takes place gradually. The patient may be able to go about for some hours after the injury, perhaps with some headache, and then he slowly loses consciousness.

Not uncommonly the loss of consciousness is preceded by other well-marked symptoms, such as headache, giddiness and vomiting. Convulsions may be a marked feature, and may be chiefly on one side. Weakness of the limbs and mental symptoms may also be present. Rupture of a large aneurysm will cause severe apoplectic symptoms.

Prognosis.—If severe coma supervenes, the prognosis is very grave. Recovery of consciousness is a good sign.

Treatment.—This is similar in the main to that of intracerebral hæmorrhage, but in traumatic cases the skull should be trephined, the blood-clot turned out, and an attempt made to secure the torn vessel or to arrest the bleeding in some other way.

vii. Cerebral Diplegia

(Congenital or Infantile Spastic Paralysis. Spastic Diplegia. Spastic Cerebral Paraplegia. Little's Disease).

Etiology.—The expression cerebral diplegia is applied to a group of diseases which depend upon widespread sym-

metrical changes in the nerve cells of the cerebral cortex. In about a third of the cases no cause can be recognised, either in the child or in the parents. Congenital tendency is sometimes a cause, since the disease may occur in more than one member of a family, and even in more than one generation. Under these circumstances there is obviously a lack of developmental power, or a tendency to premature decay, on the part of the neurons. Another cause is illness of, or injury to, the mother during pregnancy. A difficult, protracted, or otherwise abnormal labour is an important cause which operates by causing meningeal hæmorrhage.[1] Acute disease in early extra-uterine life may lead to degeneration of the neurons, and it is possible that marasmic thrombosis in veins on the surface of the brain may also be a cause. The disease is almost equally common in the two sexes.

Morbid Anatomy.—In cases where the child dies shortly after birth with convulsions, rigidity, and paralysis, meningeal hæmorrhage is found ; this may be over the motor area or in the posterior fossa. In the great majority of cases which have lived to present during life the symptoms of any variety of cerebral diplegia, the anatomical condition is one of symmetrical atrophic sclerosis. The part of the hemispheres involved varies with the clinical type. If the symptoms were chiefly mental, the frontal and occipital regions will be found to have suffered most. In paraplegic rigidity (*Little's disease*) the motor areas for the legs are at fault. In generalised rigidity with idiocy the whole cortex of the hemispheres may be involved. In bilateral athetosis the change is in the motor region, but is not so marked as in generalised rigidity.

The affected portion of the brain is shrunken ; its convolutions are irregular, and separated by wide sulci ; and there is a compensatory excess of fluid in the membranes. Microscopical examination shows extensive degeneration and loss of nerve cells (the primary process), with secondary or accompanying interstitial overgrowth. The pyramidal

[1] When meningeal hæmorrhage occurs in forceps cases, it should be regarded as due, not to the instrument, but to the abnormal labour which rendered the instrument necessary.

tract may be ill developed or degenerated. The blood-vessels are almost always healthy, but in a few cases there is symmetrical disease of the bloodvessels (thrombosis of veins, or thrombosis or embolism of the middle cerebral arteries).

Symptoms.—In many cases clumsiness or rigidity of movement is noted soon after birth. In cases of meningeal hæmorrhage there may be convulsions, asphyxia, a tense fontanelle, and other evidences of increased intracranial pressure. Paraplegic rigidity may be first noticed when the child begins to walk. Cases which are the result of acute disease may present themselves at any period of childhood. The family type usually begins between the sixth year and puberty.

In *generalised rigidity* the lower limbs usually suffer most. If the patient can walk, there is pes equinus, and there may be cross-legged progression. The face may be slightly affected. The knee-jerks are exaggerated, and the plantar reflex is extensor in type. The rigidity of the legs may render sitting impossible. Athetoid or choreiform movements may be present.

In *paraplegic rigidity*, or *Little's disease*, the variety which is specially related to abnormal labour, the phenomena are almost confined to the lower limbs.

In *bilateral athetosis* the rigidity and weakness are not so conspicuous as the constant irregular, involuntary movements. Instead of being comparatively slow or athetoid, the movements may be quick or choreoid (*choreic diplegia*).

Any degree of mental defect may be met with in cerebral diplegia. Speech may be very defective, or may never be acquired, or, having been acquired before the diplegia set in, may be lost. Convulsions are common in cases with generalised rigidity. Nystagmus, squint, inequality of the pupils, optic atrophy, dysphagia, kyphosis with falling forwards of the head, and intention-tremor are other phenomena which may be observed in cerebral diplegia.

Diagnosis.—*Paraplegia from spinal caries* may simulate paraplegic rigidity; but in the former case there may be deformity or tenderness of the spine, and a progressive increase in the weakness; while in the latter there may be

slight athetoid movements of the arms, or possibly convulsions, which will indicate the true nature of the disease.

Insular sclerosis has frequently been the clinical diagnosis in cases which proved after death to be cerebral diplegia. In the hereditary type of diplegia especially, which begins in later childhood, there may be nystagmus, changes of articulation, and optic atrophy, in addition to exaggeration of the deep reflexes and rigidity of the limbs. In a few cases, indeed, both anatomical and clinical examination have shown that the two diseases may be combined. A family tendency, however, and a very gradual onset favour the diagnosis of cerebral diplegia.

Prognosis.—Cases of paraplegic rigidity and some cases of generalised rigidity show no tendency to become worse, and may, indeed, improve under proper treatment. But many cases of cerebral diplegia show a progressive downward tendency, which leads to death after some years, from exhaustion, or from pneumonia induced by the entrance of food into the air-passages.

Treatment. — This consists in educating the mental faculties as far as possible, training the muscles so as to increase their voluntary power, passive movements, systematic rubbing, and, if need be, correction of deformities. In cases seen within a few days after birth with recurring convulsions and other pretty clear evidence of meningeal hæmorrhage, it is perhaps the best practice to trephine and remove the clot.

viii. Amaurotic Family Idiocy

(Infantile Cerebral Degeneration. Agenesia Corticalis).

This is a variety of cerebral diplegia which, so far as is yet known, is almost confined to the Jewish race. It frequently occurs in several members of a family, begins about the age of three months, and usually causes death within two years. Weakness begins in the muscles of the neck, and spreads over the trunk and limbs, being at first flaccid and afterwards spastic. Mental deterioration accom-

panies the muscular weakness, and optic atrophy and blindness also supervene. A distinctive feature of the disease is seen in the macular region, where there is a greyish-white area about twice the size of the disc, with the cherry-red macula in its centre.

The anatomical changes include intense degeneration in the neurons of the cerebral cortex, and especially in those of the motor area. The axons in the crossed and direct pyramidal tracts of the cord share in the degeneration, and the ganglion cells of the retinæ, and the optic nerves, are also involved.

ix. Cerebral Softening from Arterial Obstruction.

A cerebral artery may be obstructed by a plug brought from a distance (embolism), or by a plug formed at the spot (thrombosis). Thrombosis is often a result of local disease of the artery, though it may be promoted by an altered state of the blood, or by enfeeblement of the heart.

Embolism.—The most common cause of cerebral embolism is mitral stenosis, the plug coming not so often from the diseased valve as from a thrombus in the dilated auricle. Recurring endocarditis in a damaged valve is apt to cause embolism. Other sources are the aortic valve, the lungs, and an aortic aneurysm. The middle cerebral artery and its branches are the most common seats of embolism, and those of the left side are involved rather more frequently than those of the right, because the blood flows from the aorta into the left carotid more directly than into the right.

Thrombosis. — This occurs chiefly in connection with atheroma and syphilitic arteritis. It is favoured by changes in the quality of the blood, and by enfeeblement of the heart's action. Atheroma roughens the lining of the vessel and favours the deposition of fibrin. Syphilis narrows the lumen, and a thrombus is apt to complete the obstruction.

Morbid Anatomy.—Arterial obstruction leads to anæmia, necrosis and softening. The capillaries may or may not be distended with blood from the veins, so that the infarction may be either hæmorrhagic or pale. After twenty-four hours, the necrosed tissue rapidly softens. The colour of

the softening which results (red, yellow or white) depends upon the amount of blood present. Red softening becomes pale with time, through changes in and removal of the blood pigment. A large softening may ultimately give place to a cyst, as after hæmorrhage ; a small one may be replaced by a cicatrix. If the embolus is septic, an abscess may be produced. If death takes place soon after the obstruction occurs, the brain tissue may have a normal appearance, though it will probably feel softer to the finger than the surrounding tissue. Softening, like hæmorrhage, leads to secondary degeneration in the nerve tracts which are severed. Thus, if the internal capsule is destroyed, the pyramidal tract will degenerate from the seat of lesion to its lower termination.

Symptoms.—The symptoms of softening are in the main similar to those of hæmorrhage, and in a large proportion of cases include both general and localising phenomena ; but if the same portion of the brain is involved, hæmorrhage gives rise to more formidable consequences than softening. It may be that laceration of brain tissue causes more profound disturbance than mere deprivation of blood ; but the principal reason is the increase of intracranial pressure which is caused by intracranial hæmorrhage. As the cranium is practically a closed cavity, this increase tells on the cortex as well as on parts nearer the seat of lesion.

Softening from thrombosis may exist without symptoms.

Premonitory symptoms are absent in embolism. They are common in atheroma in the form of giddiness, headache, impairment of memory, unilateral tingling or numbness, etc. ; and are due to the imperfection of the blood-supply which is afforded by the diseased arteries. They are common in syphilis, especially in the form of severe headache, unilateral or bilateral, and worse at night.

General symptoms are not so marked as in hæmorrhage. Thus in syphilis, consciousness is generally preserved. Sudden obstruction by embolism often causes loss of consciousness. Atheromatous thrombosis in a large vessel may cause well-marked apoplexy.

The *local* symptoms consist of motor and sensory paralyses,

which vary according to the vessel obstructed. The most common palsy is hemiplegia, since the *middle cerebral* is most frequently plugged. With right-sided hemiplegia, aphasia is often associated. Complete occlusion of the *basilar* may cause bilateral paralysis of the limbs, anarthria, dysphagia, and great disturbance of temperature. The level of temperature may fall and then rise to hyperpyrexia. At the onset there may be apoplexy.

Diagnosis.—The sudden onset of hemiplegia points to a vascular lesion, viz., either obstruction or rupture. The converse is not always true, for vascular obstruction by thrombosis, especially if connected with atheroma, sometimes sets in gradually, although an onset originally described as gradual may be found on careful inquiry to have really consisted of a series of slight sudden attacks, each of which aggravated the pre-existing condition. Moreover, aphasia may set in with an abruptness which is almost sudden, and yet it may not be due to a vascular lesion. This may occur in meningitis, where the disease causes irritative inhibition of the speech centre.

In *embolism*, a possible source of an embolus is generally present. The patient usually has heart disease, or has recently had some disease, such as rheumatism, chorea, or scarlet fever, which may be associated with endocarditis. Moreover, there may be evidence of embolism elsewhere—*e.g.*. splenic enlargement with pain in the region of the spleen ; albuminuria or hæmaturia ; embolism of the central artery of the retina, etc.

In *syphilis* there is often a history of venereal infection ; severe headache may have preceded the onset ; and the age is often below forty. In young adults, almost all cases of sudden hemiplegia are due either to embolism or to syphilis.

In *atheroma* there may be premonitory symptoms, such as headache, giddiness, and numbness ; the general vigour of the nervous and circulatory organs may have been depressed by worry, overwork, or recent illness ; the onset is often gradual, or in a series of small attacks ; and the age is generally above forty. Indeed, unless there is kidney disease, or a tendency to premature arterial degeneration,

softening from atheroma is not common below the age of sixty.

In patients over forty years of age, in whom either atheromatous thrombosis or hæmorrhage is likely, the diagnosis may be very difficult. A pulse of high tension, a strongly-acting heart, hypertrophy of the left ventricle, thickening and rigidity of the arteries, and albuminuria, all favour hæmorrhage; a pulse of low tension, a feeble heart and deformity of the arteries suggest softening. Excitement may bring on hæmorrhage; exhaustion and anxiety promote thrombosis. Well-marked premonitory symptoms suggest thrombosis. Severe and prolonged coma favours hæmorrhage. Great disturbance of temperature (apart from basilar obstruction) makes hæmorrhage likely. Fatal cases of apoplexy in persons between forty and sixty almost always turn out at the autopsy to be due to hæmorrhage. The course of the illness may occasionally throw light on the case. Thus hæmorrhage occasionally occurs in young subjects from rupture of an aneurysm of one of the larger cerebral arteries, but this form of hæmorrhage is not survived. Accordingly, if a young adult recovers from an attack of apoplexy, the latter is not due to rupture of one of these large aneurysms.

Prognosis.—Basilar obstruction is almost always fatal. Thrombosis of the internal carotid artery is very dangerous to life, as the arterial disease is apt to be extensive, and thrombosis may spread widely. Embolism of the internal carotid is not so serious. The danger is much less in embolism and syphilis than in atheroma, as the patients are generally young, and disease of the arterial walls is slight or removable by treatment. The immediate prognosis will also depend largely on the general symptoms present after the onset.

Recurrence is likely in atheroma, unlikely in embolism, and unlikely, with proper treatment, in syphilis.

Treatment.—The treatment of apoplexy resulting from obstruction of a cerebral artery is similar in part to the treatment of apoplexy due to hæmorrhage (p. 766); but depleting measures must not be employed, and it may be

desirable to give digitalis or ammonia to increase the strength of the heart's action. Quiet, light diet, and careful nursing are of the utmost importance until the period of inflammatory reaction is well past. Later on tonics may be given, and the paralysed limbs may be gently rubbed. In atheroma, the patient must avoid mental and bodily exhaustion, worry and all depressing conditions. Syphilitic subjects should immediately be treated with potassium iodide (15 grains thrice daily), and this should be continued for several weeks. Syrup of the iodide of iron may be added as a tonic. These patients should, for several years afterwards, take a course of the potassium salt for two or three weeks every six months.

x. Thrombosis in Cerebral Veins and Sinuses.

Sinus thrombosis may be primary or secondary.

(1) *Primary* or *simple* thrombosis occurs chiefly in conditions of great debility, and is therefore termed *marasmic* or *marantic* thrombosis. The blood coagulates too readily, or the circulation is enfeebled, or both conditions are present. It is most common in infants, but it also occurs in adults, and especially in the old and feeble. It is chiefly seen in the first six months of life as a consequence of protracted and severe diarrhœa. It occurs in adults in the late stages of phthisis, cancer and other severe cachectic conditions, in gout, in the puerperal state, and in chlorosis. The thrombus almost always forms in the superior longitudinal sinus.

(2) *Secondary* or *infective* thrombosis is much more common, and is due to neighbouring disease. The most common cause is disease of the middle ear. Disease of some other part of the skull, injury to the skull, meningitis, and erysipelas of the head are also causes. The lateral sinus is most frequently involved.

Morbid Anatomy.—In primary cases, the wall of the sinus is seldom inflamed. The vessel is filled with adherent clot In secondary cases, the wall is often inflamed, and the clot may be softened and pus-like ; meningitis is often present. In either case, the obstruction in the sinus causes great

distension of the tributary veins, and the clot may spread into these vessels. Congestion, œdema, and sometimes hæmorrhage ensue in the brain tissue.

Symptoms.—(1) In primary cases, there may be no symptoms, and when symptoms are present they may be indistinguishable from those due to the cause of the thrombosis. The principal cerebral symptoms are stupor, coma, delirium, headache, vomiting, strabismus, muscular rigidity, and convulsions. There may be œdema and distension of veins on the side of the head, epistaxis, and in children bulging of the fontanelle. The cerebral symptoms are by no means characteristic. The œdema and venous engorgement externally are due to the obstruction to the circulation in the veins passing from the outside of the skull through the bone to the sinus. While important in diagnosis if they are present, these phenomena are seldom to be recognised. Some of the symptoms connected with thrombosis of particular sinuses are the following :

Superior Longitudinal Sinus.—There may be œdema and distension of veins on both sides of the head. There may be bleeding from the nose from veins which communicate with the sinus. The anterior fontanelle may become prominent in children, probably after being depressed in connection with the diarrhœa which led to thrombosis If the cerebral symptoms, such as convulsions, are unilateral, this suggests that the thrombus has extended into the veins over one hemisphere.

Cavernous Sinus.—There may be protrusion of the eye (*proptosis*) owing to distension of the vessels in the orbit. Œdema and venous engorgement may be present in the eyelids and neighbouring parts of the head. There may be slight swelling of the optic disc.

Lateral Sinus.—There may be œdema and distension of veins in the mastoid region. As this condition is almost always secondary to ear disease, the presence of the latter is important in diagnosis. The cerebral symptoms are suggestive of local meningitis.

(2) In secondary thrombosis, the principal phenomena are symptoms of septic poisoning, such as rigors and marked

oscillations of temperature, with symptoms and signs of pyæmic lesions in the lungs. The cerebral symptoms which are present are generally referable to the associated meningitis. There may be œdema and venous distension over the mastoid region.

Diagnosis.—This may be very difficult. The supervention of head symptoms in a case where a recognised cause of thrombosis is present should certainly suggest the occurrence of that complication, but the latter is only certain when local distension of veins or œdema is also present. The condition known as *hydrocephaloid*, which may be induced by severe diarrhœa in young children, is associated with stupor, but the fontanelle is depressed.

Prognosis.—Primary cases seldom recover. Secondary cases are hopeless unless surgical measures are adopted.

Treatment.—The primary condition should be rectified as far as possible. Secondary cases must be dealt with by the surgeon ; the thrombosed sinus may be cleared out after ligature of the jugular vein, and the primary focus should be removed altogether.

xi. Infantile Hemiplegia

(Acute Cerebral Palsy of Children. Cerebral Spastic Hemiplegia).

Etiology.—Two-thirds of the cases occur in the first three years of life ;[1] a few take origin before birth. The sexes suffer almost equally. As a rule, no obvious cause is recognisable, but in about one-third of the cases the disease occurs as a complication of some other affection, usually scarlet fever or measles. There is frequently a history of a blow on the head or of a shock immediately before the onset.

Morbid Anatomy.—Of a large number of cases analysed by Osler, a majority showed (1) atrophy and sclerosis of the

[1] Though the disease is rare after the sixth year, a similar affection is occasionally met with in older subjects. I have recorded a case occurring in later childhood and another in adolescence (*British Medical Journal*, May, 1905).

brain. These may involve groups of convolutions, a lobe, or a hemisphere. The atrophy and sclerosis are specially liable to involve the motor area, viz., the cortical distribution of the middle cerebral artery. They are associated with degeneration of the pyramidal tracts, and often with dilatation of the lateral ventricle on the affected side. The meninges are usually adherent over the atrophic portion. In about half as many cases, there was (2) *porencephalus*, which consists in the presence of cavities at the surface of the brain, and is to be regarded as one of the final stages of atrophy. The cavities appear to take origin as depressions of the surface, and may extend far into the interior of the brain; they often communicate with the ventricle. They are lined by pia mater, and bridged over by arachnoid. They contain subarachnoid fluid, and may thus resemble cysts. Porencephalus is doubtless in many cases congenital. In a still smaller proportion of cases there was (3) embolism, thrombosis, or hæmorrhage. In this small group the children were usually a good deal older than in the other cases.

In recent encephalitis, the affected portion of the hemisphere is purple in colour, and the convolutions are swollen. It is chiefly the grey cortex which is involved.

Pathology.—Many of the congenital cases are due to syphilis in the mother, the lesion in the child being syphilitic disease and thrombosis in the middle cerebral artery or in veins on the surface of the hemisphere. In rare cases the same kind of lesion has been found when the disease set in some time after birth. In a few cases the cause of the hemiplegia may be injury to the head, or meningeal hæmorrhage, at the time of birth. The majority of all cases, however, are due to *encephalitis* (*polioencephalitis*), a condition which in some ways is very similar to poliomyelitis. Both are doubtless of infective nature, and it is indeed possible that the virus is the same in both cases. It has happened that of two members of a family who became ill at the same time, the one suffered from poliomyelitis, and the other from polioencephalitis. Even when the hemiplegia occurs in the course of another infection, such

as scarlet fever or measles, encephalitis appears to be the cause in the majority of cases, while in the remainder a vascular lesion, such as thrombosis or embolism, is probable. According to James Taylor, the different morbid processes arranged in order of frequency are : (1) acute encephalitis, (2) thrombosis, arterial or venous, (3) hæmorrhage, and (4) embolism.

Symptoms.—In the majority of cases, the onset is attended by severe convulsions. These are generally unilateral, and involve the limbs which are afterwards found to be paralysed. The child may be unconscious for hours or days, and there may be pyrexia and vomiting. Right hemiplegia in children who have learned to speak may be accompanied by aphasia, but this passes off before long. The voluntary movement of the face suffers less than emotional movement (in laughing, crying, etc).[1] Sensation is seldom affected, and never permanently. The permanent loss of power may be slight. If the loss is complete at first, some recovery takes place after a time, but the upper limb very often remains seriously weakened. In this limb there are often developed the phenomena of *mobile spasm*, *athetosis*, or *post-hemiplegic chorea* (see pp. 759, 760). The digits are constantly being flexed and extended, usually by slow movements ; and adduction, abduction, pronation, and supination may also be observed in the different parts of the limb. The affected limbs do not grow so well as the others, but the patient becomes able to walk. The knee-jerk is exaggerated. Mental defect is a common result, and may manifest any degree of severity. Another common sequel is recurring convulsions, which involve, as a rule, only the paralysed limbs.

Diagnosis.—In most of these cases there is a distinct attack of acute head symptoms, setting in some time after birth. Hemiplegia of chronic onset is due to tumour.

[1] This is different from what obtains in the most common form of hemiplegia in adults, and has repeatedly been observed in cases of infantile hemiplegia where the lesion was found on anatomical examination to be confined to the motor cortex. See note on p. 755.

Prognosis.—In some cases the paralysis disappears completely in the course of a few months, but even then there may be some residual mental impairment, and epilepsy may supervene about the period of puberty. But in many cases the hemiplegia, with or without mental impairment and athetosis, proves to be permanent, and in addition there may be recurring convulsions.

Treatment.—Convulsions at the onset should be treated by bromides given by the rectum, and by chloroform inhalations. Later on, the paralysed limbs should be treated by systematic rubbing and passive movement. Deformities should be prevented as far as possible, or corrected—if need be, by operation—if they are already in existence. Recurring convulsions should be treated as if they were due to ordinary epilepsy.

xii. Insular Sclerosis.

(Disseminated or Multiple Sclerosis).[1]

Three varieties of this disease are recognised : cerebral, spinal and cerebro-spinal. The last is the usual form.

Etiology.—This is obscure. The disease is occasionally a sequel of an acute infection, pregnancy or parturition, mental distress, injury, or exposure ; but in a large proportion of cases no cause can be recognised. The disease occurs chiefly in early adult life, and is rare after fifty. It affects the two sexes almost equally.

Morbid Anatomy.—Reddish-grey patches of sclerosis are scattered at random throughout the central nervous system and sometimes also in the cranial nerves. They generally develop in the white substance. In the brain they are common in the centrum ovale, central ganglia, crus cerebri and pons, but they seldom invade the cortex. In the cord they favour the lateral columns. Their size varies up to that of a walnut. Their shape is irregular. Their margin is well defined. Their colour is slightly darker than that of the normal grey cortex. Their consistence is slightly, and occasionally a good deal, firmer than that of normal brain tissue.

[1] French, *Sclérose en plaques disseminées.*

The patches are characterised by overgrowth of neuroglia, thickening of the walls of the vessels, and wasting of the nerve fibres. But the white substance of the fibres wastes first, and the axis cylinders may persist for a long time. Occasionally there is secondary degeneration. Insular sclerosis thus differs from the system or neuron degenerations in the random distribution of the lesions.

Pathology.—According to some, the interstitial overgrowth is primary and the degeneration of nerve fibres secondary, while others would reverse this order. It has been suggested that the interstitial change results from thrombosis of minute vessels, and that the vascular changes which lead to the thrombosis may be due to some toxic agent.

Symptoms.—The symptoms vary greatly in different cases. (1) One of the most frequent is muscular weakness of varying distribution, often partial, and often of sudden origin. Indeed, one of the most common modes of onset of the disease is with sudden, incomplete loss of power in one limb. The limb is not altogether disabled, but the patient lets articles drop if the upper limb is at fault, or drags the foot a little if the leg is involved. Some numbness or tingling may be associated with the loss of power, and a noteworthy fact is that after a time the power may be regained. Paresis may afterwards return in the same or in another limb. Weakness in the legs is almost always associated with spasticity and exaggerated knee-jerks. Ankle clonus may be present, and the plantar reflex is extensor in type.

(2) Another very striking symptom is coarse, jerky irregular movement, frequently involving the hand, and observed only in connection with voluntary movement (*intention-tremor*). For instance, if the patient tries to convey a cup of water from the table to his mouth, the limb becomes the seat of such violent jerking that the water is quickly spilled. Yet when no voluntary movement is attempted, the limb is usually perfectly still. In the course of the disease, similar tremors may involve the muscles of the trunk, neck, face, tongue and legs. In the early stages, the tremor may be slight, and may then be seen when the patient writes. This form of inco-ordination is supposed to depend upon the non-uniform retardation of

impulses passing along the different efferent fibres at the sclerosed patch; it is believed to coincide with sclerosis of the pons. Sclerosis in the posterior columns may give rise to ataxy in the legs.

(3) Eye symptoms are common, and include (*a*) impaired vision in one or both eyes, with contraction of the fields, but without, at first, ophthalmoscopic changes. Impairment of vision in one eye may be the first symptom, and it may, like paresis of a limb, pass away after a time, to return later on in the same or in the opposite eye. The symptom is attributable to sclerosis in the optic nerves or chiasm, and this leads to secondary atrophy. There may, however, be primary optic atrophy, exactly as in tabes dorsalis. (*b*) Nystagmus is a common symptom, but the oscillating movements are usually observed only in connection with voluntary movement of the eyes. (*c*) There may be external ocular palsies, involving especially convergence and conjugate deviation. Transient weakness of an ocular muscle causing squint is sometimes the first symptom of the disease. (*d*) Internal palsies are uncommon, but any form may be met with.

(4) Articulation is sometimes *scanning* (*staccato*, *syllabic*), or *elisive* (*slurring*), or both; the syllables being separated and accentuated in the first variety, and the ends of words being elided in the second. These symptoms point to involvement of the bulbar region.

(5) Cutaneous sensibility may be impaired, often in an irregular and localised way. There may be paræsthesiæ, sharp or dull pains, and girdle sensation. There may be dissociated anæsthesia, one form of sensation being lost while others are preserved. Thus in one case there was loss of the stereognostic sense in the right hand, the patient being unable to recognise the shapes of objects, although the senses of touch, pain, and temperature were preserved.[1] Sensation, however, is often normal.

(6) There may be some delay in evacuation of the bladder, and there may be irritability of that viscus, and even incontinence.

[1] *Transactions of the Glasgow Pathological and Clinical Society*, February 11, 1901.

(7) Mental change is common, but is generally slight. The most common manifestation is abnormal contentment in spite of increasing disability. But the patient may become childish, unduly emotional, and even demented ; or there may be mania, melancholia, or delusions of grandeur recalling those of general paralysis.

(8) Occasionally there are paroxysms of headache, vertigo and vomiting.

(9) Sometimes there are epileptiform or apoplectiform seizures, characterised by coma, convulsions, hemiplegia, and pyrexia.

Trophic lesions of the skin, muscular wasting, and changes in the electrical reactions of muscles are not phenomena of this disease.

Diagnosis.—The intention-tremor, the progressive weakness of spastic type, the nystagmus and the change in the articulation, are sufficiently characteristic if they are all present.

In *paralysis agitans*, the head is bent forwards, the features are fixed, the muscles are rigid, and the commencement is after forty ; but above all, the rhythmical tremor continues when no movement is willed. Nystagmus is never present.

In *general paralysis of the insane*, tremor is most marked in the lips and tongue, and mental symptoms are present.

Mercurial tremors may resemble those of insular sclerosis exactly. The history and occupation of the patient should be sufficient for diagnosis.

Insular sclerosis is apt to be mistaken for *hysteria*. In insular sclerosis, loss of power and of sensation are commonly less complete than in hysteria. Moreover, the partial loss of power may be transient, and may recur in the same or in another limb, a state of matters not observed in hysteria. Transient paresis of external ocular muscles is not seen in hysteria. Blurring or considerable impairment of vision, transient, and recurring in the same or the other eye, seldom occurs in hysteria. Optic atrophy is conclusive evidence against mere functional disorder. Persistent and well-marked ankle clonus suggests sclerosis of the pyramidal tract ; and the extensor type of plantar reflex (Babinski's

sign) points to organic change. In hysteria, the plantar reflex is often absent, and when present, is of flexor type. Nystagmus, intention-tremor and scanning articulation are not present in hysteria.

Prognosis.—The disease is apt to progress by fits and starts, and great temporary improvement may take place. The duration may be a few or many years. The prognosis of optic atrophy in this disease is not nearly so grave as in tabes. Death may be due to interference with deglutition or respiration, or to intercurrent disease.

Treatment.—Arsenic, silver nitrate, and small doses of mercury and potassium iodide may be tried, but no cure is known.

xiii. Hydrocephalus

(Dropsy of the Brain. Water in the Head).

Hydrocephalus consists in an abnormal accumulation of fluid within the skull. It may be *acute* or *chronic*, *primary* or *secondary*, *external* (*subdural*) or *internal* (*intraventricular*), *congenital* or *acquired*.

Acute Hydrocephalus is always due to meningitis, and indeed acute hydrocephalus is now synonymous with tubercular meningitis. It may be external, or internal, or both.

Chronic External Hydrocephalus is usually a compensatory condition, the fluid under the dura occupying the space left by senile shrinking, or by imperfect growth of the brain. In some cases, however, without undue smallness of the brain, there is a congenital excess of subdural fluid, and this causes the head to enlarge. A living birth may be impossible, but if this is accomplished, the skull rapidly expands. The clinical features are then similar to those of internal hydrocephalus.

Chronic Internal Hydrocephalus may be congenital or acquired.

(1) Of the *congenital* form, no certain cause is known. It may occur in several members of a family. It may advance so far in intra-uterine life as to make living birth im-

possible, or it may develop shortly after birth or even at a later period.

Morbid Anatomy.—The lateral ventricles are greatly distended by a watery fluid, with the result that the brain tissue around them is thinned by stretching. The foramen of Monro may be greatly widened. The middle ventricle is also enlarged, and sometimes the iter and fourth ventricle. The fluid is not unlike dropsical fluid. It contains albumen and saline matter, and has a specific gravity ranging some degrees above or below 1010. Not merely is the convexity of the hemisphere thinned by the distension of its cavity, but the convolutions tend to become flattened and unfolded. The lining membrane of the ventricle is thickened. The corpus callosum shares in the stretching and thinning, and it occasionally ruptures during life, so that the fluid escapes from the ventricles, and the brain collapses. The optic tracts may be much damaged by pressure. The fontanelles are enlarged, and the cranial bones separate at the sutures, expand and become thin. In later stages, if the patient lives, and the disease becomes arrested, the gaps between the bones may be partly filled up by the development of Wormian bones or *ossa triquetra*. The cranium is enlarged out of all proportion to the face, so that the eyes look downwards. The vertex of the head is unduly convex, and contrasts with that of rickets, which is unduly flat.

Symptoms.—The gradual enlargement of the head is usually the first sign of the disease, and may indeed go on for many months before any other symptoms supervene. In other cases, the enlargement is preceded by a period characterised by fretfulness of disposition and perhaps squinting and convulsions. The enlargement involves the cranium and causes the face to appear small in proportion. The latter is often old-looking and devoid of expression. The summit of the head is rounded, the skin over it thin, and the hair scanty. Fluctuation can sometimes be detected. Mental defect is generally present. There is sluggishness of intellect, with loss of memory and a tendency to sleep much by day, or the defect may amount to

complete idiocy. As the disease advances, the head is imperfectly supported, partly on account of its weight, and partly on account of the muscular weakness. The limbs become weak, and may become the seat of contractures as well as convulsions. Blindness from optic atrophy may result from the pressure on or stretching of the optic nerves, tracts and chiasma. The eyes are turned downwards, and rolled from side to side.

Prognosis.—Many cases die within a few months after birth, from coma, convulsions, debility, or some intercurrent disease. A number, however, survive for one or several years. Occasionally the morbid process undergoes arrest, and the patient may live to advanced life, though probably the subject, throughout life, of mental irritability and weakness, and sometimes of epileptic fits.

(2) The *acquired* form sometimes occurs without discoverable cause, and has been regarded as primary or spontaneous, but this is doubtful. It is generally secondary to pressure (usually by a tumour) on the veins of Galen, to closure (by meningitis) of the foramen of Magendie and the other openings by which the cerebro-spinal fluid escapes from the fourth ventricle, or to obstruction of the iter by a tumour or by parasites. The cerebro-spinal fluid is secreted to a large extent by the choroid plexuses of the lateral ventricles, and then passes through the third ventricle and Sylvian aqueduct to the fourth ventricle, to reach the openings in the pia mater. Obstruction of the aqueduct will cause the fluid to accumulate in the middle and lateral ventricles. Compression of the veins of Galen will lead to a dropsical accumulation in the same cavities. Meningitis (*e.g.*, of the posterior basic variety, or secondary to a tumour) may, by obliterating the openings in the pia mater, lead to accumulation in the fourth, as well as in the third and lateral ventricles. Tumours which obstruct the aqueduct and the veins of Galen are chiefly those which grow in the structures underneath the tentorium cerebelli, viz., in the cerebellum, pons and corpora quadrigemina.

Symptoms.—These are in the main similar to those of the congenital disease, and include mental and muscular weak-

ness, blindness, convulsions and coma. Before the sight is completely lost, there may be bitemporal hemianopia, in consequence of pressure on the middle part of the chiasm. In young children, the head may enlarge greatly, but in adults this is exceptional, and unless there is enlargement, the diagnosis cannot be made with certainty. Any symptoms that may be present may be masked by those of tumour, if such is the primary lesion.

Prognosis.—The condition usually terminates fatally within a few months or years.

Treatment.—No satisfactory measures are available for the treatment of chronic hydrocephalus. Repeated lumbar puncture should have a fair trial. Compression of the head by strips of plaster, or by a broad elastic band, may be employed for several periods of a week each. The intervals between these periods should likewise be of a week's duration, and may be occupied with inunction of mercurial ointment into the shaven scalp. Attempts have been made in recent years to establish permanent drainage of the ventricles by making a communication between the latter and the subarachnoid or subdural space. Bromides should be given for convulsions. In cases of tubercular tumour, cod-liver oil, syrup of the iodide of iron, and country or seaside air are indicated.

xiv. Inflammation of the Brain (Encephalitis. Cerebritis).

Acute Encephalitis.

Etiology.—Acute inflammation of the brain may be the result of injury to the head—*e.g.*, a blow, fracture, punctured wound, or simple concussion. The inflammation is usually just beneath the seat of injury, but occasionally develops at the opposite side as a consequence of *contrecoup*. The meninges often suffer with the brain, as might be expected, but occasionally the latter alone is involved ; and, moreover, the inflammation may not quite reach the surface. The cerebritis may subside, or may pass on to abscess formation. Another cause is disease of the skull, whether in connection with the ear, nose or orbit,

or brought about in other parts of the skull by trauma or by syphilis. Meningitis is usually present in addition, and the cerebritis generally proceeds to suppuration.

Cerebritis is sometimes observed in connection with acute infections such as erysipelas, diphtheria, influenza, typhus, and enteric fever. It is well seen in cerebro-spinal meningitis. Alcoholism and other intoxications are also causes. Localised cerebritis may be due to septic embolism. The acute encephalitis which causes many cases of infantile hemiplegia has been already described (pp. 779-782).

Acute inflammation occasionally involves the nuclei of the nerves of the ocular muscles (*polioencephalitis superior acuta*), or the nuclei of the bulbar nerves (*polioencephalitis inferior acuta*), just as it involves the anterior horn of the spinal cord in infantile paralysis (*poliomyelitis anterior acuta*).

Morbid Anatomy.—The inflammation may be disseminated, and characterised by multiple minute foci, as in erysipelas and diphtheria; or focal, as in septic embolism, though this too may be multiple; or confined to grey matter, as in polioencephalitis. The anatomical condition of an acutely inflamed part is that known as *red softening*. The affected tissue is softened and swollen, so that on section it is elevated above the surrounding brain substance. Its limits are not well defined. The redness is due partly to injection of its small vessels, but still more to minute hæmorrhages. The nerve cells, nerve fibres and neuroglia cells of the part all undergo degeneration and give rise to granular cells. Leucocytes are present in excess. The softening gives rise to a kind of emulsion stained with blood pigment, and the ultimate result may resemble that which follows necrotic softening from vascular obstruction.

In the disseminated inflammation of specific infections, the foci may be characterised principally by accumulations of round cells, with or without microbes.

Symptoms.—The general symptoms include headache, and sometimes vomiting, delirium and general convulsions. Optic neuritis is rarely observed. Localising symptoms vary according to the seat of lesion. The most important are weakness and convulsions in the limbs on the side oppo-

site to the lesion. As Gowers remarks, these symptoms are not uncommon a few days after a head injury, and may be accompanied by pyrexia; whereas if they were due directly to injury to the brain, they would set in immediately, and if they were due to abscess, they would not develop for some weeks after the injury.

The symptoms of cerebritis occurring in the course of specific fevers are not always distinguishable from those due to the toxic blood state.

Acute superior polioencephalitis is characterised by the acute development of ophthalmoplegia, viz., paralysis of the motor ocular nerves.

Acute inferior polioencephalitis shows itself by the acute development of bulbar paralysis.

Diagnosis.—The symptoms may be practically the same as those of meningitis over the same portion of brain; and they may also resemble those of any toxic blood state which may be the cause of the inflammation. The diagnosis may thus present great difficulty.

Prognosis.—Actual recovery of the inflamed brain tissue can only be expected when the lesion is very slight. The outlook in any particular case must be inferred from the severity of the symptoms.

Treatment.—Rest in bed, purgation, and the application of cold or of leeches to the head, are the principal remedies.

Cerebral Abscess.

Etiology.—The most common cause of cerebral abscess is chronic suppurative otitis media. The ear disease has usually existed for years, sometimes for many years, and in many cases the abscess follows arrest of the discharge. The bone is generally but not always carious. The abscess generally develops in the temporal lobe of the cerebrum, much less frequently in the cerebellar hemisphere. The infection may reach the brain by direct extension, by the perivascular lymphatic sheaths, by perineural lymphatics, or by the medium of septic thrombosis in veins. Cerebral abscess is generally due to disease of the tympanum, because

the roof of the latter constitutes part of the middle fossa of the skull on which the temporal lobe lies, and both send their blood to the superior petrosal sinus. Similarly, disease of the mastoid cells tends to cause cerebellar abscess, because the roof of the mastoid antrum is part of the posterior fossa on which the cerebellar hemisphere rests, and both send their blood to the lateral sinus.

Injuries of the head are another cause of cerebral abscess, the latter being most common in the frontal and parietal regions. The injury may be a blow causing fracture or necrosis, or it may be a punctured wound.

Disease of the skull, apart from the temporal bone, and apart from injury, is an occasional cause of cerebral abscess. It may be syphilitic, tubercular or cancerous, and is occasionally connected with the nose or orbit.

In a fourth group of cases, cerebral abscess is metastatic. The primary disease is generally in the lung, and as a rule there is not general pyæmia. In about half of these cases, the cerebral abscess is multiple. This kind of abscess (*pulmonal cerebral abscess*) does not occur in ordinary phthisis, but may be met with in empyema, bronchiectasis, pulmonary gangrene, etc. Cerebral abscess may also arise in connection with acute fevers such as influenza or enteric.

Males suffer from cerebral abscess far more frequently than females. All ages are liable, but more than half the cases are met with between ten and thirty.

The septic infection may be due to almost any pyogenic organism, or may be mixed.

Morbid Anatomy.—The abscess is usually solitary, and is frequently surrounded by a capsule. The pus is greenish, and has sometimes a fœtid odour. If connected with a fracture of the bone, the abscess is generally superficial; but in cases of injury to the head without fracture, and in cases due to ear disease, it is usually situated deeply, being separated from the surface by normal brain tissue. When an abscess has become surrounded by a thick capsule, it may remain stationary for a long time. Such an abscess is more or less rounded in form. It may be of minute size, or may appear to occupy more than half of the hemisphere.

Often, however, before any thick capsule forms, the abscess tends to grow and cause death, either by exerting pressure, or by bursting into a lateral ventricle or (less commonly) on to the surface of the brain. The consequence of bursting is suppurative inflammation of the lining membrane of the ventricle, or of the meninges, as the case may be. An abscess due to bone disease may communicate with the bone through the membranes, and may even discharge in this way through the ear or other part.

Symptoms.—The abscess may be acute or chronic. (1) In *acute abscess*, which is usually due to local injury or to infection from a distance, the symptoms resemble those of meningitis, which, moreover, is often present. They include intense headache, vomiting, rigors, and fever at the outset; and after a time, delirium, convulsions, paralysis on the side opposite the abscess, and fatal coma. The usual duration is from one to four weeks.

(2) In *chronic abscess*, the early symptoms may be slight and may be unnoticed. After a time, there may be a latent period of months or years during which the abscess is practically stationary, and symptoms are few or altogether absent. During this period, in ear cases, headache may alternate with otorrhœa. Then the terminal stage sets in, gradually or suddenly, with symptoms similar to the later phenomena of the acute form. The general symptoms are more important than the focal. Thus if the cranial nerves are paralysed, it is generally through associated meningitis or bone disease.

There is often severe headache limited to one side. Vomiting, drowsiness, optic neuritis, a slow pulse and constipation may be present. The temperature may be mostly subnormal, but occasionally disturbed by a sudden rise accompanied by rigor, and followed by sweating.

Localising phenomena are occasionally present. Thus *abscess in the temporal lobe* may cause symptoms of pressure on the internal capsule, or on the third nerve of the same side. If the abscess is in the left temporal lobe, it may cause word-deafness. *Cerebellar abscess* causes severe vomiting and optic neuritis, with nystagmus on move-

ment of the eyes, disturbance of equilibrium, and occasionally weakness of the limbs and exaggeration of the knee-jerk on the side of lesion. It may also cause retraction of the head, or inclination of the head to the side of lesion. It may cause 'skew deviation' of the eyes (the eye on the side of lesion being rotated outwards and upwards, and the other eye outwards and perhaps downwards). Later on, there may be deviation of both eyes away from the side of lesion. *Abscess in or near the motor area* may produce localised weakness or spasm of muscles, and perhaps motor aphasia.

Diagnosis.—In acute cases, where injury to the head, the constitutional symptoms of suppuration, and symptoms of intracranial disease are all present, there is no room for doubt. In chronic ear disease, the supervention of any head symptom must be regarded with great suspicion. But in chronic ear disease, fever and head symptoms may be due to other lesions than cerebral abscess—*e.g.*, meningitis, sinus thrombosis or mastoid disease. *Abscess* gives rise to increasing somnolence, a normal or subnormal temperature, a slow pulse, and possibly signs of a focal lesion in the temporal lobe or some other part of the brain. *Meningitis* causes increased irritability, rigidity, twitchings of the muscles, fever, a rapid pulse, delirium, and signs of interference with cranial nerves. In *sinus thrombosis*, there are pain and tenderness in the region of the jugular vein, the vessel may be hard, there may be rigors and fever as in pyæmia, and the pulse is rapid; but the intellect is clear, and there are no focal brain symptoms. In *mastoid disease* the intellect is clear; the mastoid region is the seat of pain, tenderness and perhaps swelling; pyrexia is present, and the pulse is accelerated; but there are no focal brain symptoms.

When chronic head symptoms are present, a diagnosis must be made between *tumour* and abscess. The existence of a possible cause such as ear disease, injury, or suppuration elsewhere, favours abscess. If ear disease is not admitted by the patient or the friends, it is all the more necessary that the ears should be examined. Coexistent

pulmonary tuberculosis is in favour of tumour but not of abscess. Fever, rigors and an acute onset of symptoms favour abscess. Malignant disease elsewhere points to tumour.

Prognosis.—Unless the symptoms have been completely latent for a very prolonged period, the case is practically hopeless unless surgical measures are adopted.

Treatment.—Preventive treatment is of great importance. Wounds about the head must be kept aseptic, and no ear should be allowed to continue suppurating for an indefinite period. When symptoms of abscess set in, surgical measures must be promptly resorted to; and in cases secondary to injury or to ear disease, there is good ground for hope that the patient's life may be saved. Apart from surgical measures, the treatment is symptomatic.

xv. Intracranial Tumours.

Etiology.—Males suffer from cerebral tumour more frequently than females. Brain tumours may occur at any age, but are not common in old persons. Half of the tubercular cases occur in the first ten years of life. Glioma and sarcoma are met with especially in the active period of adult life. In tubercular cases, there is often a family history of phthisis, and if the patient is an adult he is frequently the subject of that ailment. According to Gowers, syphilitic growths are most common from five to twelve years after infection. In rare instances a brain tumour—*e.g.*, the teratoma—is of congenital origin. In a certain number of cases, an injury to the head appears to be the cause of tumour, and with regard to the common tubercular growth, this would be quite in accordance with what we know of tubercular disease in other parts of the body. But it must be remembered that a blow on the head may cause hæmorrhage in an already existing tumour.

Setting aside syphilitic tumours, Gowers estimates that more than half of the cases of cerebral tumour are tubercular, and that glioma and sarcoma together constitute a third.

Cancer of the brain is almost always secondary. Cysts

may result from degenerative changes in such tumours as the glioma and sarcoma. Parasitic cysts are also met with, chiefly in foreign countries ; viz., *Cysticercus cellulosæ*, the bladder worm of *Tænia solium ;* and hydatid cysts, the larval stage of *T. echinococcus*. Both of these parasitic cysts doubtless arise in consequence of the eggs of the worms having been taken into the stomach.

Aneurysms of the larger cerebral arteries constitute intracranial tumours, and are specially related to syphilis and embolism. They are considered separately hereafter.

Myxoma, endothelioma, fibroma, psammoma, angioma, cholesteatoma, and other tumours are occasionally met with.

Tumours in the substance of the brain are most common in the cerebral hemispheres ; but in proportion to its bulk, the cerebellum is far more apt to suffer.

Morbid Anatomy.—The *tubercular* (*scrofulous*) *tumour* (see p. 131) is often multiple. It is almost equally common in the cerebellum and in the cerebral hemisphere. The growing margin is grey and translucent, from the presence of recent granulation tissue in the midst of which are giant cells containing tubercle bacilli. The central portion is yellow and opaque from caseous change. The ordinary size of the tumour varies from that of a pea to that of a walnut. It is generally found within the brain substance, and not obviously connected with the membranes. It compresses and destroys the brain tissue ; and as its peripheral part is soft, and the tissue around is frequently soft also, the tumour can be shelled out. This tumour is occasionally the only tubercular lesion present, but is in most cases secondary to tuberculosis elsewhere. Tubercular meningitis may be present, and acquired hydrocephalus may be a result of pressure by the growth.

The *syphiloma* (*gumma*) is common and occurs chiefly in the cerebral hemisphere and pons. It is usually superficial, taking origin from the outer wall of the bloodvessels in the soft membranes, and thence extending into the brain, doubtless by way of the perivascular sheaths. It often acquires adhesions to the dura mater also. These growths vary in size up to that of a chestnut. They are irregular

in form, and are found on section to have undergone caseation in an irregular manner ; in these two respects they are differentiated from tubercular growths.

The *glioma* takes origin in the neuroglia, infiltrates the brain, and has no well-defined margin. In consistence, and often in colour, it resembles the normal grey matter. In structure it is allied to neuroglia. It may be the seat of fatty degeneration, cyst formation, and hæmorrhage ; and the last may give rise to apoplectiform symptoms. The growth is most common in the cerebral hemispheres and cerebellum.

The *sarcoma* may begin in the brain substance, pia mater, dura mater, or bone. When it begins in the brain itself, it is well defined, and may have a distinct capsule surrounded by softened brain tissue, so that it can be enucleated. Growing from the bone, it may perforate the dura mater. Growing from the bone or from the dura mater, it may grow through the bone and cause an external swelling ; and it may compress the brain, though it is not likely to perforate the pia mater. Sarcoma of the brain may be secondary to a growth elsewhere.

Cancer is chiefly soft, and may begin in the dura mater or brain. It is usually secondary.

Symptoms.—A cerebral tumour may, by its presence and growth, irritate the cerebral tissue and cranial nerves, either directly or by causing inflammation. By compressing the brain tissue and the cranial nerves, it may cause loss of function in the parts compressed. Moreover, as it grows it is apt to cause increase of intracranial pressure, and this is likely to be aggravated if the growth is so situated as to give rise to hydrocephalus (see p. 788). The symptoms of brain tumour may thus be divided into two classes : (1) *general* or *diffuse* symptoms, corresponding in the main to the results of increased intracranial tension ; and (2) *focal* or *localising* symptoms, resulting from irritation, or from loss of function, of particular portions of the brain or particular cranial nerves

(1) The principal *general symptoms* are headache, vomiting, optic neuritis, giddiness, general convulsions, syncopal

and apoplectiform attacks, slowing of the pulse, loss of flesh and strength, polyuria, disorders of speech, and mental disturbance.

Headache is one of the earliest and most frequent symptoms. It is often of agonising severity, interfering with sleep, and increased by effort, excitement, or any agency which stimulates the cerebral circulation. It persists after delirium sets in, and occasionally, like meningitis, induces the hydrocephalic cry. It is often continuous, but subject to paroxysmal exacerbations. It may be diffuse, and even when localised in one part of the head may be due to tumour in another part. If, however, there is severe pain which can be localised to a small, well-defined and constant area, this is probably a focal symptom, and is pretty certainly so if it is accompanied by local tenderness.

Vomiting is sometimes an early symptom, and may then be misinterpreted. A hint may sometimes be obtained from the absence of any other evidence of gastric disorder, such as nausea or furring of the tongue. But nausea may be present, and the vomiting may occur specially after the taking of food. Vomiting is particularly common in cases of cerebellar tumour.

Optic neuritis is present at one period or another in most fatal cases, and is usually bilateral. It may be well marked, although the patient is unaware of any visual impairment ; but after some time, and especially if consecutive atrophy supervenes, the visual acuity may fail seriously, and even go on to complete loss of sight. The immediate cause of optic neuritis is a matter of dispute, but it is probably due largely to the intracranial pressure causing dropsy of the sheath of the nerve with compression of its vessels ; and possibly in part to inflammation of the sheath excited by irritating substances in the cerebro-spinal fluid, or by extension from meningitis. Optic neuritis is specially common in connection with tumours of the cerebellum and quadrigeminal bodies.

Giddiness is a common symptom, and may be constant or paroxysmal.

General convulsions may resemble those of ordinary

epilepsy, or may simulate hysteria. Or, again, there may be attacks of petit mal.

Syncopal and apoplectiform attacks may result from hæmorrhage into the tumour.

The speech may be abnormally slow, or the words may be cut short and run together. More definite alterations in speech may occur as focal symptoms when the bulbar region or speech centres are involved.

Mental disturbance may assume various aspects. It is not surprising that the patient, worn out by pain and want of sleep, should gradually lose some of his higher faculties, such as the power of attention and memory. He may become greatly depressed, or unduly emotional, and may suffer from hallucinations and delusions. The mental lethargy becomes such that the evacuations are passed into the bed, and the case gravitates from stupor towards fatal coma.

(2) *Focal symptoms* are not always present. Local tenderness of the skull, and a very localised pain in the same part are important symptoms when they happen to be present, and are suggestive of a growth which involves the meninges. Dilatation of surface veins will occasionally help to indicate which side of the brain is affected.

A tumour pressing upon the motor area of the cortex may cause convulsions which always begin in any given case in the same group of muscles, and may even be confined to these muscles (*Jacksonian epilepsy*). A localised sensory disturbance, such as tingling, may precede the convulsion, and has the same significance. Whenever the growth damages the brain substance, localised paralysis may be added to, or take the place of, the localised convulsions or spasm. Tumours which involve the speech centres will give rise to corresponding disturbances of speech. Tumours involving the special sense regions of the cortex may impair those special senses ; or, by irritating the centres, give rise to corresponding hallucinations (visual, auditory, etc.).

Tumours in the corpus striatum may interfere with the internal capsule and cause hemiplegia. Tumours of the optic thalamus may interfere with the posterior end of the

internal capsule, and may thus cause hemianæsthesia, as well as hemianopia and athetosis.

Tumours of the frontal lobe may interfere with the olfactory bulb and tract and so impair smell. Mental dulness and even imbecility may occur early.

Tumours of the cerebellum may cause a reeling gait, nystagmus, weakness of the body on the side of lesion, loss or exaggeration of the knee-jerk, occipital headache, vomiting, and rigidity of the neck. There may be attacks of rigidity as in tetanus, with retraction of the head. Optic neuritis is early and severe. Chronic hydrocephalus may be caused by a tumour of the middle lobe. Localising symptoms may be few, because in chronic disease of the cerebellum compensation readily takes place.

Tumour of the pituitary body is in some cases, but not in all, accompanied by the phenomena of *acromegaly*. It may give rise to bitemporal hemianopia and ultimately blindness (see p. 638).

Tumours of the corpora quadrigemina, pons and bulb are likely to damage the nuclei and roots of cranial nerves and thus produce localising symptoms. Tumours of the crus cerebri, pons and bulb cause *alternate hemiplegia*. Tumours of the pons and bulb may involve both sides, and thus cause *double hemiplegia*. Growths in the quadrigeminal bodies are likely to involve the oculomotor nucleus ; growths in the pons, the fifth, sixth, and seventh nuclei ; and growths in the medulla, the nuclei of the cranial nerves from the eighth to the twelfth.

Tumours at the base of the brain are apt to interfere with the cranial nerves. Different disturbances of sight are produced by involvement of the visual path in the optic nerve, optic chiasm, and optic tract. The third nerve may be involved by tumours in or near the crus cerebri, and both third nerves may be paralysed by one tumour situated between the two crura. The fifth nerve suffers especially from tumours which involve the middle fossa of the base of the skull. The sixth nerve is often paralysed, on one or both sides, by tumours situated underneath the tentorium cerebelli, which, as they grow, compress the nerve between

the brain and the base of the skull. Tumours of the posterior fossa may damage the cranial nerves from the fifth to the twelfth.

Internal hydrocephalus may be produced by a growth situated underneath the tentorium cerebelli, or even as far forward as the third ventricle.

It must be remembered in practice, first, that cerebral tumours may give rise to no symptoms,[1] and, secondly, that they are frequently multiple.

Diagnosis.—Headache, vomiting and optic neuritis are the cardinal symptoms of intracranial tumour, and if the neuritis is intense, this combination of symptoms is seldom due to anything except tumour. The addition of a focal symptom such as paralysis of a cranial nerve makes the diagnosis practically certain. The combination of headache, vomiting and optic neuritis is not very uncommon in *Bright's disease;* but in that case, the neuritis is not intense, changes are pretty sure to be found in the macular region of the retina, and albumen and casts will be found in the urine. Headache and optic neuritis may also be met with in severe *anæmia* and in *lead poisoning*, but the concomitant facts of each case should make the diagnosis clear to a careful investigator. The diagnosis from *cerebral abscess* has been considered in the account of that disease.

The situation of the tumour is inferred from the localising symptoms, but in many cases this cannot be done with any degree of certainty.

The nature of the tumour can sometimes be inferred with considerable probability. Thus if a malignant growth is present in another part of the body, the intracranial tumour is likely to be of the same nature. Brain tumours occurring in syphilitic and tubercular subjects are presumably due to

[1] In the case of the late Professor John Hughes Bennett, who died after lithotomy, the autopsy revealed a soft tumour, about as large as a hen's egg, between the dura mater and the bone, a little above the right ear. The growth fitted into a deep hollow in the brain, and over part of its extent the parietal bone was completely replaced by fibrous membrane. Bennett had known of the depression in the skull from childhood. There were no cerebral symptoms. (See *Brit. Med. Journ.*, 1875, ii., 453, 454.)

the respective infections. As Gowers points out, a patient under fifteen, with no signs of inherited syphilis, does not suffer from a syphilitic growth ; and an adult, with no signs of phthisis, is not at all likely to have a tubercular tumour in his head. Cortical tumours are often syphilitic. Syphilitic tumours are frequently removable by antisyphilitic treatment.

Prognosis.—A favourable prognosis can be given with considerable confidence only in the case of syphilitic growths, and even these may leave permanent damage behind them. Tubercular growths not uncommonly become stationary, and other tumours may at times undergo prolonged or permanent arrest. Thus I have met with a case in which the left lobe of the cerebellum was almost replaced by a myxoma which caused severe symptoms for about a year at the age of sixteen, and then became latent. With the exception of blindness from consecutive optic atrophy, and an attack of typhus at twenty-nine, the patient enjoyed good health for about forty-five years, after which he died of cancer of the pylorus at the age of sixty-three.[1]

Cancers and soft sarcomas grow quickly. Gliomas, on the other hand, may grow very slowly. In non-syphilitic cases, death generally results within two years. Death may be due to syphilitic or tubercular lesions of another kind ; to exhaustion ; or to coma and other results of increased intracranial pressure. Sometimes it takes place suddenly.

Severe optic neuritis, even though the growth which induces it is removed by treatment, passes into consecutive or post-neuritic atrophy with permanent impairment or loss of vision.

Treatment.—In syphilis, iodide of potassium and mercury are indicated. Even in non-syphilitic cases benefit may occasionally arise from antisyphilitic treatment. In tuberculosis, cod-liver oil, syrup of the iodide of iron, and country or seaside air should be tried. Analgesics and hypnotics will often be called for to relieve pain and permit of sleep. Large doses of potassium iodide, the ice-bag, and even the

[1] *Transactions of the Glasgow Medico-Chirurgical Society*, April 3, 1896.

actual cautery, may be tried for their analgesic effects. In a small proportion of cases, the growth can be removed by operation. In cases where the disease cannot be extirpated, operation is sometimes advisable to relieve pressure symptoms, such as severe headache, vomiting, or optic neuritis.

xvi. Aneurysms of the Larger Cerebral Arteries.

Etiology.—These aneurysms are more common in males than in females. They are equally common before and after forty years of age (thus differing from miliary aneurysms), and may occur at any age between ten and sixty. Embolism which does not produce complete occlusion of the vessel is a common cause, since the artery, damaged and perhaps inflamed, is still exposed to the pressure of the blood. Very old syphilitic arteritis may lead to aneurysm, especially of the basilar artery. Injury to the head may cause it, especially in the internal carotid artery.

Morbid Anatomy.—The aneurysm varies in size, and may become as large as a walnut. In its growth, it may become completely embedded in the brain substance. When it ruptures, the hæmorrhage usually takes place into the membranes at the base, but it may take place only into the brain substance. In a number of cases, however, the aneurysm does not rupture. The middle cerebral artery, being the most frequent seat of embolism, is the most frequent seat of this disease.

Symptoms.—The disease may be completely latent. The embolism which causes the aneurysm generally produces no symptoms.

When symptoms of aneurysm are present, headache, which is often pulsating, and giddiness are among the most common. There is often palsy of cranial nerves, and sometimes palsy of the limbs. It is quite exceptional to hear a murmur. The case generally ends by rupture of the aneurysm, with an always fatal result.

Diagnosis.—This is difficult and often impossible. The condition might be suspected if there were symptoms of a tumour at the base of the brain in a patient with heart

disease. In a syphilitic case, failure of specific treatment would be suggestive.

Treatment.—If the existence of an aneurysm is recognised, the patient should live a quiet life, and avoid severe strain on the vascular system. The treatment recommended for aortic aneurysm may be tried. If the aneurysm is pretty certainly connected with the internal carotid, the common carotid should be ligatured.

FUNCTIONAL AND NUTRITIONAL DISEASES OF THE NERVOUS SYSTEM.

i. Chorea

(Sydenham's Chorea. Chorea Minor. St. Vitus's Dance).[1]

Etiology.—Four-fifths of the cases occur between five and fifteen years of age ; very few after twenty. Girls suffer from two to three times as often as boys, and after the twentieth year the liability is almost confined to females. Heredity has some influence, in respect of both rheumatic and neurotic tendencies.

An association with rheumatism as manifested by arthritis or pains is present in many cases ; the proportion is put by different writers at from a sixth to a half of the cases. The frequency tends to increase as the child grows older. Either disease may come first.

Heart disease, and especially endocarditis, is also related to chorea. Here again either affection may come first. Heart disease is more common in recurrences of chorea than in first attacks—a point of resemblance between

[1] The designation St. Vitus's dance (*chorea Sancti Viti*) was first applied at Strasburg some five hundred years ago to an epidemic dancing mania, for the cure of which the sufferers were sent by the Strasburg authorities to the chapel of St. Vitus, at Zabern, an Alsatian village in the neighbourhood. The name is the only connecting link between the dancing mania and what is now called *chorea*. The Germans, however, apply the expression *chorea major* to a hysterical disorder of movement, which they distinguish from *chorea minor*, or chorea in the modern acceptation of the word.

chorea and rheumatism. Chorea, like heart disease, may be associated with scarlatinal rheumatism.

Chorea sometimes develops in pregnancy, usually without any other recognisable cause. It is most common in the first pregnancy, and may or may not recur in a later one. It begins most commonly in the third month, but may set in at any stage.

The only recognised immediate cause of chorea is fright or some other strong emotion. This is operative in about a fifth of the cases, the symptoms generally setting in several days after the fright. Fright seems to be the exciting cause in about the same proportion of cases of chorea in pregnancy as of cases in early life. Fright is also a cause of relapse in a certain proportion of cases of ordinary chorea.

Morbid Anatomy.—There is no constant lesion of the nervous system. Embolisms are sometimes found in the small cerebral arteries, and these were at one time regarded as the cause of the disease. The organisms of rheumatism have been found close to the cells of the cerebral cortex. The heart is diseased in the great majority of fatal cases. There is endocarditis, either old or recent, most commonly involving the mitral valve; and pericarditis is not uncommon.

Pathology.—The theory which appears to be most closely in accordance with known facts is that chorea is one of the numerous manifestations of the rheumatic diathesis. A rheumatic tendency or actual infection by the rheumatic poison, a neurotic tendency, and a strong emotion appear to be, when acting in conjunction, the most likely agents to cause chorea. But it is quite possible that chorea may, like endocarditis, pleurisy, erythema and other inflammations, be caused by other agencies besides the specific microbe or toxin of rheumatic fever. The special liability of late childhood, and of girls at that period, and the frequent coexistence of endocarditis and other rheumatic lesions, are in accordance with the rheumatic theory.

The motor symptoms are to be attributed to a disturbance of function in the grey matter of the motor area of the cerebral cortex. This disturbance may be due either to

the general toxic state, or to the organisms acting locally upon the nerve elements.

Symptoms.—These include (1) spontaneous movements which are irregular in time and degree. They involve the face, tongue, trunk and limbs, and are increased by excitement and by voluntary movement. They cease during sleep, though they are occasionally so violent as to prevent sleep. Chorea has been called 'insanity of the muscles.' In exceptional cases, the patient may be seriously hurt, or even thrown out of bed, by the violent jerkings of her limbs and trunk. The movements are sometimes confined to, and often most marked on one side of the body (*hemichorea*). The arms suffer sooner and more than the legs. The movements are commonly most marked in the fingers, and may be such as to prevent the patient from balancing an article on the extended open hand. She has the appearance, when sitting upright, of being very restless, and the face is the seat of grimaces. Similar movements may be observed in the toes, and the respiratory muscles may be observed to contract suddenly and irregularly.

(2) Inco-ordination is present, so that the patient lets articles fall from the hand, spills her tea, and is unable to keep the tongue protruded for more than a few seconds.

(3) The affected muscles are weak, and occasionally weakness is almost the only symptom present (*paralytic* or *paretic chorea*). In a few instances (*chorea mollis*, *limp chorea*) the paralysis is severe and involves the whole body. It may or may not be preceded by the usual symptoms of chorea. Quite exceptionally, muscular rigidity, with or without pain, may be the most obtrusive feature (*spastic chorea*).[1]

(4) Speech is often impaired, owing to involvement of the muscles of articulation and respiration, and possibly also to involvement of the larynx. Speech is apt to be uttered quickly and to be interrupted. The patient is often disinclined to speak, owing doubtless to the difficulty, and perhaps also through slight mental change.

(5) Mental disorder is sometimes present. It is usually slight, and is manifested by irritability of temper, dis-

[1] *Glasgow Medical Journal*, 1897. xlvii., 95.

obedience to parents, etc. Sometimes, however, the mental deterioration is so great that the evacuations are passed into the bed, or there may be maniacal excitement (*maniacal chorea, chorea insaniens*).

(6) The temperature is normal in slight cases, but may be elevated in severe attacks.

(7) Various abnormalities may be recognised in connection with the heart. Its action is often accelerated, and may be irregular owing to the disturbance of the respiratory movements. The left ventricle may be dilated. Hæmic murmurs may be present in consequence of the anæmia. Murmurs and other evidence of valvular disease are common, especially in older children. The most common murmur is mitral systolic.

There is seldom any marked sensory disturbance, though the patient sometimes complains of pain in the limbs. This, if not rheumatic, is probably due to fatigue of the constantly active muscles. The electrical irritability of the muscles and nerves is sometimes increased both to faradism and to galvanism.

The knee-jerk is often unduly sustained. Thus in a patient with hemichorea, the healthy limb promptly falls, after being jerked up, whilst on the affected side, the leg remains elevated for an appreciable period before falling.

Other manifestations of rheumatism may be present, such as pericarditis, pleurisy, arthritis, subcutaneous nodules, erythema marginatum, tonsillitis, and hæmatoporphyrinuria.

Sequels.—In rare cases, bodily weakness (*post-choreic paralysis*) or mental feebleness may persist for some time after an attack of chorea. Patients may also suffer for a time from sudden starting of the limbs, which may even cause them to fall.

Diagnosis.—*Huntington's chorea*, or *hereditary progressive chorea*, in which the involuntary moments are exactly similar to those of chorea, begins later in life than ordinary chorea. It is hereditary, progressive, and incurable, and it is associated with progressive mental deterioration.

Senile chorea also begins, as a rule, in the degenerative period of life.

Electrical chorea, or *Dubini's disease*, is met with in Northern Italy, and is characterised by sudden muscular contractions, such as might result from electrical stimulation. It is associated with muscular weakness and wasting.

Post-hemiplegic chorea is a disorder of movement which occasionally follows an attack of hemiplegia.

Certain forms of *cerebral diplegia*[1] have been described as 'congenital chorea.' But cerebral diplegia is often detected within the first three years of life, is frequently progressive, and may be associated with signs of organic disease, such as paralysis, rigidity, and recurring convulsions.

In *hysterical chorea* the movements are more rhythmical than in ordinary chorea. Other evidences of hysteria may be present.

Prognosis.—From a few weeks to a few months is the usual duration of an attack. Recurrences are common, and may, like first attacks, be due to fright. Chorea almost always ends in complete recovery, but occasionally it persists as a chronic disease. The mortality of the chorea of childhood is about 2 per cent., and is due to exhaustion, heart disease, hyperpyrexia, pyæmia (resulting from pyogenic infection of abrasions due to the movements), or intercurrent disease. Death occurs chiefly in first attacks, and in individuals who have reached the period of puberty. The chorea of pregnancy is ten times as fatal, though even here death is due to abortion, complications of labour, etc., rather than to the nervous disease itself.

Treatment.—Mental and physical rest must be secured. Even in mild cases, the child should be kept in bed at first. In severe cases, a water-bed should be used, and the patient must be watched in case of injury through the violence of the movements. If these prevent sleep, chloral should be given at the outset as a sedative and hypnotic. As much fattening food should be given as the patient can utilise. If swallowing is difficult, the child should be fed by the

[1] Namely, bilateral athetosis, diplegia with choreic movements, diplegia with myoclonic movements, and diplegia with intention-tremor (James Taylor, 'Paralysis and Other Diseases of the Nervous System in Childhood and Early Life,' p. 254).

nasal tube, the latter being lubricated with olive oil, and not with glycerin. A full meal given in this way, and containing a dose of alcohol, may act as an efficient hypnotic. A daily warm bath is helpful; the patient should sit in the bath and have tepid or even, after a time, cold water poured over her shoulders. The procedure ought to be grateful to the child, and no shock must be administered.

One of the best drugs is arsenic, which should be given in moderate doses (2 to 5 minims of Fowler's solution thrice daily, after meals); large doses should be avoided. Antipyrin sometimes gives good results. Sulphate of zinc is another remedy; 3 grains may be given to begin with, but the dose should be gradually increased. Strychnine also may be employed. Lees advocates large doses of sodium salicylate (100 grains daily for a child of from six to ten years; the dose to be rapidly increased to 150 or even 200 grains) combined with twice the quantity of sodium bicarbonate.

Eustace Smith recommends for children of all ages 1 drachm of liquid extract of ergot, diluted, every three or four hours, with complete rest in bed. For intractable cases Taylor recommends hyoscine hydrobromide ($\frac{1}{100}$ grain hypodermically, thrice daily); but this drug should not be used in acute cases, or in cases associated with heart disease or emaciation. Another mode of treatment is to keep the patient constantly asleep for several weeks by means of chloral.

Iron is indicated for anæmia. When the patient recovers, care should be taken that she is not overworked at lessons.

ii. Chronic Adult Chorea.

Huntington's Chorea[1] (*hereditary progressive chorea*) occurs in families and sometimes affects several generations in succession. The two sexes are equally liable. The disease generally begins between thirty-five and fifty, and affects first the face and upper limbs. Articulation and

[1] Described by Huntington of New York in 1872, but previously described by older writers.

deglutition may be disordered. The gait may become unsteady, and ultimately all the muscles may be involved. The movements resemble those of chorea, but are slower. There is generally mental impairment, which goes on to dementia. The scanty post-mortem evidence seems to indicate that the disease is a chronic meningo-encephalitis leading to atrophy of the convolutions.

Diagnosis.—This affection is differentiated from ordinary chorea by the hereditary tendency, late onset, and associated mental symptoms. Pathologically the two diseases seem to be entirely different.

Prognosis.—The disease is incurable, but the fatal issue may be postponed for many years.

Senile Chorea differs from Huntington's chorea in not running in families and in not usually being associated with mental symptoms. In some cases, moreover, the disease may be cured, though it generally persists till death. In most cases it has no relation to rheumatism. It seldom begins before fifty-five, but a very severe case was under my care in a man aged thirty-eight. Speech was involved, the patient was unable to feed himself, and for a time deglutition was embarrassed. Considerable improvement took place under treatment by hydrobromide of hyoscine.

iii. Electrical Chorea (Dubini's Disease).[1]

Electrical chorea is a rare disease which is met with in Northern Italy, and is characterised by sudden muscular contractions such as might result from electrical stimulation. There is progressive paralysis and wasting of the muscles. Both sexes and all ages are liable.

The sudden contractions generally involve one arm at first, and afterwards extend to the leg of the same side, and at a later period to the opposite limbs. Loss of faradic irritability accompanies the weakness and wasting. Epileptiform convulsions and pyrexia may also be observed.

The disease often terminates fatally within a few months or even weeks.

[1] First described by Dubini, an Italian physician, in 1846.

The disease is supposed to be an infection. No constant anatomical changes have been found, and no satisfactory treatment is known.

iv. Myoclonus (Paramyoclonus Multiplex).

This is a rare disease affecting males more frequently than females, and setting in at almost any period of adult life. It may develop spontaneously, or after a fright, or after some other affection. In some instances it has attacked several members of a family. The disease is often complicated with epilepsy.

Pathology.—According to different theories, the anterior horn cells of the cord, the cerebral cortex, or both upper and lower motor neurons are at fault.

Symptoms.—The disease is characterised by sudden shock-like contractions in symmetrical muscles of the limbs, and sometimes of the trunk. The distal portions of the limbs, and the face usually escape. In some cases the contractions are not sufficient to produce displacement of the limbs, while in other cases they are. Excitement and fatigue tend to increase the spasm ; voluntary movements may diminish or aggravate them ; sleep arrests them. The muscles do not waste, and there is no involvement of sensation or of the sphincters. Some cases show slight mental defect, and many are epileptic.

The term *myokimia* has been applied to a variety of this disease in which the muscular fibres involved in each contraction are so few that the condition resembles the fibrillary twitchings seen in chronic spinal muscular atrophy.

Prognosis.—The symptoms sometimes diminish considerably with the lapse of time, and recovery may occur, but the disease is apt to prove very intractable.

Treatment.—No successful treatment is known, but nervine tonics such as strychnine and arsenic, and sedatives such as bromides and hyoscine, are clearly indicated. Galvanisation of the spine and of the affected muscles, and hydropathic measures, have been reported upon favourably. Epilepsy must be treated by a bromide.

v. Impulsive Tic

(Maladie des Tics. Gilles de la Tourette's Disease).

This disease, distinct on the one hand from the convulsive tic or facial spasm of advanced life, and on the other hand from the simple habit spasm of childhood, is characterised by mental as well as by motor symptoms. It generally begins in childhood, and there is often a neurotic family tendency. The symptoms may be first evoked by some distinct cause, such as mental shock. The involuntary muscular twitchings commonly begin in the orbicularis palpebrarum, and tend to spread down the face to the neck and sometimes to the arms. After a time there may be involuntary ejaculations, mimicry of words (*echolalia*), mimicry of actions (*echokinesis*), use of bad language (*coprolalia*), and imperative ideas. Recovery may occur, but the outlook is not very hopeful.

vi. Habit Spasm

(Simple Tic. Habit Chorea).

This affection is most common in later childhood, though sometimes seen in adults. It is characterised by habitual movements which the patient can control for a limited period of time by an effort of will, but which recur as soon as his attention is withdrawn. He is rendered uncomfortable by trying to control them. The condition is generally recognised as a trick, or bad habit. Among the common movements are winking, drawing the mouth to one side, shaking the head, shrugging the shoulders, sniffing with the nose, and biting the lips or the finger-nails. These habits generally die out as the patient grows, but they sometimes persist, and may cause much annoyance. They are doubtless often inaugurated by some local cause. Thus sniffing of the nose may have its origin in adenoids and nasal catarrh, and winking in ophthalmia or errors of refraction.

Treatment.—The general health should be seen to, and any local disorders corrected. In the case of children, a reward may be promised for each day which is kept entirely free from the movements. If the sufferer is an adult.

arsenic, bromides, strychnine, and possibly local blistering may be tried. Massage and gymnastics may also be recommended.

vii. Head Nodding

(Spasmus Nutans. Salaam Spasm).

This affection is met with chiefly in the first year of life. The child is usually in good general health, and the cause of the spasm is obscure, although it has been suggested that living in a badly lighted room may have some influence. It is assumed that the phenomena depend upon some instability of the motor cortex.

The most common movement of the head is from side to side. Occasionally it is a nodding movement. In a few cases the whole trunk is bent forward as if to make a salaam. Nystagmus is often present, and may be either lateral or rotatory. Sometimes there is momentary loss of consciousness, but there is seldom any mental defect.

The prognosis is perfectly favourable. Bromides, belladonna, cod-liver oil, and other tonics are the remedies indicated.

viii. Eclampsia Nutans

(Salaam Convulsions).

This affection is met with in the period between the first dentition and puberty. It is characterised by bowing movements of the trunk, which may be so extensive that the head approaches the knees. Paroxysms occur in which the patient goes through many such movements, and many of these paroxysms may take place in one day. There may be momentary loss of consciousness. Mental enfeeblement frequently supervenes, but some cases recover. The treatment is similar to that recommended for head nodding.

ix. Saltatory Spasm.

In this condition there are clonic contractions of the muscles of the legs when the patient stands, so that in slight cases the heels are raised and let down in quick

succession, while in severe cases there are jumping movements. Both men and women have suffered from this rare disease, which is apparently a pure neurosis. Recovery frequently takes place, but the affection sometimes persists for years. The principal indication is to rectify any underlying neurasthenic or hysterical condition.

x. Paralysis Agitans

(Shaking Palsy. Parkinson's Disease).[1]

Etiology.—The disease is one of later life, and is rarely seen before forty. Men suffer more frequently than women. In most cases there is no obvious cause, but anxiety, mental shock, physical injury, exposure, and acute disease are occasional causes.

Morbid Anatomy. — No constant anatomical lesion is known. Any changes observed are such as might be found in any old person.

Pathology.—The symptoms are probably due to impaired nutrition and disordered function in the motor area of the cortex. As Gowers points out, the cessation of the tremor during sleep, the unilateral commencement and the hemiplegic mode of extension make it unlikely that the spinal cord is the primary seat of disturbance. Further, as the symptoms are at first purely motor, and as the first cells met with in the motor path above the level of the cord are those of the motor area of the cortex, it seems probable that they, or the grey matter in which their dendrons take origin, are at fault. This view is strongly corroborated by the fact that an attack of ordinary hemiplegia arrests the tremor in the paralysed limbs. In chorea, which is also supposed to be due to disorder of the motor region of the cortex, certain analogous phenomena are observed, including spontaneous movement, diminished power of movement, cessation of the movements during sleep, and frequently a unilateral distribution.

Symptoms.—(1) Tremor is generally the first symptom.

[1] First fully described by James Parkinson, an English physician, in 1817.

It usually begins insidiously in the hand, spreads gradually to the rest of that limb, and then to the leg of the same side. It next invades the opposite arm, and finally the opposite leg.

Much less commonly a leg is first affected. The disease then usually spreads to the arm of the same side, and then to the opposite arm, but occasionally it spreads from the leg to the opposite leg.

The tremor usually confines itself to the limbs, but occasionally involves the muscles of the back and neck. Of course the trunk and head may shake owing to tremor transmitted from the limbs. The shaking is rhythmical in character, and is due to alternating contractions of opposing muscles. It is most marked in the hands and fingers, where it is due to the action both of the long muscles and of the intrinsic muscles of the hands. In the legs the calf muscles are specially liable to be affected. The muscles which act on the jaw and the tongue are occasionally affected ; the muscles of the face very seldom.

The tremor is fine in the early stages, but increases in range as the disease advances. According to Gowers, its rhythm varies from 4·8 to 7 complete oscillations per second. It almost invariably ceases during sleep, but it almost always continues during rest when the patient is awake. It is increased by mental excitement, and is often temporarily arrested by a voluntary movement.

(2) Weakness and (3) rigidity generally set in together after the tremor. Voluntary movements are slow as well as weak.

(4) The attitude is characteristic, and is attributable to the muscular rigidity. The head and neck are thrown forwards and fixed. The features are fixed and mask-like. The face has an anxious expression. The elbows are flexed. The fingers are often in the interosseal position (flexion of metacarpo-phalangeal, and extension of interphalangeal joints). The hips and knees are flexed, and the thighs are adducted.

(5) The gait is also altered. The patient may have difficulty in rising from his seat, and once he has started to walk, he takes short quick steps as if running (*festination* or

propulsion). If he is gently pulled by the coat from behind, he may be started to walk backwards, and may then be unable to stop until he is arrested by some obstacle such as the wall of the room (*retropulsion*).

(6) Speech may be slow and difficult, or it may be slow to start, and then hurried like the gait. Difficulty of deglutition may be associated with the difficulty in articulation.

There is little or no change in the reflexes, muscular nutrition or activity of the sphincters. It is noteworthy that nystagmus is never observed in this disease. The principal sensory disturbance is an abnormal sense of, or sensibility to heat, and this is often accompanied by an abnormal readiness to perspire.

Diagnosis.—The first five symptoms enumerated above are characteristic. The tremor is absent in a few cases, but this of itself does not invalidate the diagnosis.

In *insular sclerosis*, the tremor occurs only on movement, and is usually coarse and irregular. The disease generally begins before forty years of age. Nystagmus is often present. The attitude and gait of paralysis agitans are absent, and the speech defect is different.

In *mercurial tremors*, the occupation of the patient explains the condition.

Prognosis.—The disease is chronic, and as a rule slowly progressive during many years. It does not directly cause death.

Treatment.—No cure is known. The patient should avoid mental and physical strain. Malt extract and cod-liver oil may improve the general condition. Arsenic, strychnine, phosphorus, iron and cannabis indica may be tried. To relieve the tremor, the affected parts may be rubbed, and the patient should take hydrobromide of hyoscine ($\frac{1}{50}$ grain or more in chloroform water) two or three times a day.

xi. Various Forms of Tremor.

Tremor occurs under many different circumstances in health and disease. It may be a transient phenomenon resulting from exposure to cold, or from severe emotional excitement. In certain diseases it may accompany volun-

tary movement (*intention-tremor*), as in insular sclerosis, and in some cases of tumour (especially tubercle) of the pons or crus. In another group the tremor goes on continuously as long as the patient is awake, as in paralysis agitans. In other diseases, again, the tremor may be seen either with voluntary movement only, or independently of it ; or it may occur along with voluntary movement in an early stage, and become continuous in a later stage. To this extensive group belong chronic poisoning by alcohol, tobacco, opium, chloral, lead, and mercury (*toxic tremor*) ; the typhoid state ; exophthalmic goitre ; hysteria ; neurasthenia ; general paralysis of the insane ; infantile hemiplegia ; and cerebral diplegia.

Senile tremor occurs in old persons, and affects the head early, though it usually begins in one or both arms. It may be increased, or only brought out, by voluntary movement.

Simple tremor sometimes begins at puberty or later, and then usually persists through life. It is frequently absent during rest, but is induced by movement, and aggravated by excitement. It affects chiefly the hands and head, and seldom interferes much with complex actions such as writing. It may be inherited, and it may affect several members of one family (*hereditary tremor*).

Asthenic tremor may be seen as a transient phenomenon resulting from severe exercise of certain muscles, as in the arm after carrying a heavy load. Tremor occurring only on movement is observed in conditions of feebleness such as may follow acute disease.

xii. Torticollis

(Wryneck).

In this condition, the head is fixed in an unnatural position owing to abnormal contraction of one or more muscles of the neck.[1]

[1] Torticollis must be clearly distinguished from transient *stiffneck*, which is a form of muscular rheumatism (p. 228).

Congenital Torticollis (*fixed wryneck*) depends upon shortening of one sternomastoid muscle, which is often wasted. Though usually congenital, this affection may not be noticed for some years after birth. The head cannot be rotated to the side on which the muscle is affected (usually the right side).

Etiology.—It is supposed that fixed wryneck is due in some cases to imperfect development of the affected muscle in connection with the position of the fœtal head within the uterus. In such cases, the face may be non-symmetrical. In other instances, the condition may result from injury (*e.g.*, hæmatoma) of the muscle produced during labour.

Treatment.—Tenotomy is an effectual remedy.

Spasmodic Torticollis is due to active muscular spasm, which may be tonic, or clonic, or both. It is usually confined to, or more marked on, one side.

Etiology.—The disease generally commences in middle life, and often without obvious cause; though exposure, emotion, injury, and a neurotic tendency may have some influence. There is a difference of opinion as to the relative frequency with which the two sexes suffer.

Morbid Anatomy and Pathology.—No anatomical changes are known. It is uncertain whether the overacting centres are spinal or cortical, but on the whole the evidence seems to point to derangement of the cortex. It is possible that different centres are at fault in different cases. As Gowers points out, an objection to the view that the disturbance is cortical is found in the fact that disease of the cortex which causes deviation of the head usually causes also deviation of the eyes; whereas in ordinary torticollis, the ocular muscles are never involved. On the other hand the frequently associated spasm of the splenius of one side and the sternomastoid of the other side suggests that it is not simply the lowest motor centres in the cord which are disordered. Poore says there is frequently an irritable condition of the spinal accessory nerve, but how this comes about is not clear. The same writer thinks that weakness of the muscles on one side, as from overwork, tends to excite contractions

in the imperfectly antagonised muscles of the opposite side. In any case, spasmodic wryneck is to be regarded as a pure neurosis.

Symptoms.—These set in gradually, usually in the course of months. As a rule, several muscles are involved, the sternomastoid being most constantly affected. If only one muscle is involved, it is almost always the sternomastoid. The upper part of the trapezius on the same side as the affected sternomastoid, the splenius of the opposite side, and the platysma myoides are also often at fault. Occasionally the spasm involves the arm, face or masticatory muscles.

The position of the head naturally varies according to the muscle or combination of muscles involved. It is generally turned to one side, and most commonly to the left side. The sternomastoid muscle of one side rotates the head towards the opposite side. The same sternomastoid acting with the opposite splenius causes extreme rotation. One sternomastoid with the trapezius of the same side causes a strong inclination of the head to the same side. The sternomastoid of one side with the opposite trapezius causes great rotation with little inclination.

In a few cases the muscles suffer alike on the two sides, and the head is drawn back so that the face looks upwards (*retrocollic spasm*). The spasm, whether tonic, clonic, or both tonic and clonic, varies in severity from time to time. It ceases during sleep. When the patient is alone and thinking of other matters, even severe spasm may diminish considerably; whereas excitement and the presence of strangers tend to aggravate it. As the disease progresses, the spasm becomes more severe in degree, and frequently spreads to other muscles than those which it first attacked.

Though the disease is one of motor elements, the severe spasm may give rise to a feeling of fatigue or even pain. The affected muscles tend in the long-run to become hypertrophied. Their electrical irritability is either normal or increased.

Diagnosis.—It is important not to misinterpret deviation of the head due to *disease of the cervical spine*. In this condition, the one sternomastoid is tense from being put on the

stretch ; *i.e.*, it is tense on the side towards which the face is turned.

Prognosis.—The disease may remain slight, or may become severe. It usually persists through life, and even after disappearing for a time, is liable to recur. It does not shorten life.

Treatment.—Temporary benefit is sometimes obtainable from bromides, valerianate of zinc, asafœtida, cannabis indica or conium. A better remedy is hyoscine hydrobromide ($\frac{1}{200}$ grain or more in chloroform water, thrice daily, after food). In severe cases, neurectomy is indicated. A piece of the spinal accessory nerve is cut out, and if other muscles besides the sternomastoid are at fault, the corresponding branches of the cervical nerves must be similarly dealt with.

xiii. Tetany

(Tetanilla. Idiopathic Muscular Spasm).

Etiology.—The disease is most common in little children and in the second decade of life, but is not infrequent in the first half of adult life. In infancy, it is chiefly males, and after twenty, chiefly females who suffer. It sometimes occurs in epidemic form. In most cases, there is a distinct cause, such as severe diarrhœa, exposure, pregnancy, lactation, dilatation of the stomach, acute fevers or thyroidectomy. Trousseau called the disease *nurse's contracture* from its occurrence in women who were suckling. Young children who suffer are almost always rickety.

Morbid Anatomy.—No distinctive lesions are known, but it is probable that there are at least nutritional changes in the peripheral motor neurons. Active proliferation of the parathyroid cells has been described.

Pathology.—Experimental destruction of the thyroid alone is followed by the gradual development of myxœdema ; while destruction of the parathyroids alone causes acute, rapidly fatal nervous phenomena, including tetany. Cases of tetany that follow thyroidectomy are thus accounted for by loss of the parathyroids along with the thyroid, for

it would appear that the function of the parathyroids is to neutralise some poison which is produced in the course of metabolism. The variety of conditions which give rise to tetany suggests either that a variety of poisons may produce the symptoms, or that various circumstances may lead to the generation of the metabolic poison in such quantity that the parathyroids are unable to neutralise it.

Symptoms.—The principal symptom is the occurrence of painful tonic spasm in the hands and feet. This is often preceded by tingling or numbness in the same parts. The fingers are fixed in the interosseal position (flexion of metacarpo-phalangeal and extension of interphalangeal joints) and drawn together. The thumb is adducted, and may be flexed. The position of the hand is sometimes described as ' the accoucheur's hand.' The wrist and often the elbow are slightly flexed. The ankle is extended ; the foot is strongly arched and turned in ; the toes are drawn together and flexed. The knee is usually extended. The spasm may involve the proximal parts of the limbs, and in severe cases, it may spread to the trunk and even to the muscles of the face, eyes, tongue, and larynx, and to the muscles of mastication. Involvement of the muscles of the abdomen, chest and larynx may seriously embarrass respiration. The spasm of the abdominal muscles may give rise to emprosthotonos. There may be retention of urine.

The spasm sets in suddenly. It almost always affects both sides of the body. It may be confined to the hands and feet, or even to the hands. It interferes with, and may prevent movement.

The contracture may be continuous, intermittent, or remittent in type. An attack of the continuous variety may persist for weeks. But in most cases, the condition is paroxysmal, and the attacks or exacerbations may last for minutes or hours. The intermissions or remissions may last for hours or days. Sleep may diminish, but does not always abolish, the spasm.

Important sensory phenomena are common. Numbness or burning is commonly felt in the hands and feet for some time before the spasm sets in. When the spasm is at all

severe, there is great pain in the muscles and along the nerve-trunks, and sensation may be blunted in the affected parts. Passive extension of the rigid muscles aggravates the pain. There is no loss of consciousness.

In the interval between the attacks, the muscles and nerves continue unduly irritable, as is shown by the fact that muscular contraction is called forth by percussion on the muscle or pressure on its motor nerve. This is specially well seen in the face (*Chvostek's sign, facial phenomenon*). As Trousseau pointed out, compression of the nerves or bloodvessels of the parts will call forth an attack (*Trousseau's phenomenon*); this begins in the course of a few minutes.

Both the galvanic and the faradic irritability of the nerves is much increased. Moreover, there is a change in the polar reactions of the muscles to galvanism, ACC or even AOC being obtained more readily than KCC which comes first in health.

In very severe attacks there may be pyrexia, with a rapid pulse and profuse perspiration. There may be swelling of the dorsa of the hands and feet.

Diagnosis.—This depends on the character, distribution and symmetry of the spasm. The special involvement of the hands and feet, and the absence of trismus, as well as the etiology, distinguish the disease from *tetanus*.

Prognosis.—Recovery almost always takes place in the course of a few weeks or months. There is however danger to life in cases due to dilatation of the stomach or to thyroidectomy. Moreover, the cause of the tetany, *e.g.*, diarrhœa in children, may cause death. The tetany of pregnancy usually persists until delivery takes place.

Treatment.—The cause must be removed where this is possible, as in the case of lactation. The general health must be attended to. The spasm should be allayed by bromides or chloral given internally, and by local anodyne applications. Rickety children should get cod-liver oil, chemical food, and as soon as possible country or seaside air. Thyroid extract should be tried in cases following thyroidectomy.

xiv. Occupation Neuroses.

Constantly recurring use of a particular set of voluntary muscles sometimes causes them to become the seat of irregular or imperfectly co-ordinated movements which interfere with voluntary movement. In the earlier stages, the disordered movement or cramp only interferes with the frequently repeated movement which was its cause. After a time, it may begin to interfere with other voluntary movements, and in some cases, it may even occur spontaneously. Among those who suffer from these neuroses are clerks and others who write much, telegraphists, piano-players, violin-players, drummers, sempstresses, milkmaids, cigarette-rollers, and others.[1] The most common variety is writer's cramp.

Writer's Cramp (*Scrivener's palsy*), as Gowers points out, is due to a bad mode of writing. Though the patient has generally made excessive use of his muscles in writing, the manner of writing is more important in the causation of this disease than the amount. Similarly, the mode of moving the pen is much more important than the mode of holding it. Accordingly, men who write in a very cramped fashion may suffer from and be disabled by the disease, although they actually write very little. The individual who is most likely to be attacked usually moves the pen by moving the two or three outer digits, whilst the wrist and even the little finger are fixed on the desk. Gowers also observes that professional shorthand writers, who must of necessity write in a free style, and who generally write from the shoulder, almost never suffer from this disease. In writing from the shoulder, there are slight contractions of large and powerful muscles. In writing with the wrist fixed, there are proportionately great contractions of very small muscles, which are thus overtaxed.

Anxiety, a neurotic tendency, and local injury or disease appear to favour the onset of the affection. It is much

[1] Comparable to these neuroses which involve the hands are the nystagmus of miners, and the cramp of dancers.

more common in men than in women. It usually begins between twenty and fifty years of age.

Morbid Anatomy and Pathology.—No constant morbid changes are known. The condition is one of irritable weakness of nerve centres, but whether these are cortical or spinal is uncertain.

Symptoms.—The onset is usually gradual. The patient begins to feel that he cannot write with the same ease as formerly, and his hand aches a little after his day's work. Then his handwriting begins to suffer owing to unsteadiness of his fingers ; and as he tries to hold the pen more firmly, the fingers become more readily fatigued and more unsteady. He tries one new method of holding the pen after another, and each fails in turn, until at length, if he perseveres in the attempts, the handwriting may become almost illegible through the irregular movements induced by every effort to write.

The most important symptom, then, is spasm or cramp, which sets in when the intended action is begun, and interferes with it. The spasm is almost always tonic, though occasionally clonic.

In a few cases, instead of tonic spasm, there is tremor, pain, or weakness, and the varieties of the disease in which these symptoms exist alone or predominate are styled respectively *tremulous*, *neuralgic* and *paralytic*. The *spasmodic*, *spastic* or common form is that which has been described in detail. The paralytic type is very rare. In the common form, there is sometimes diminution of power.

In cases of writer's cramp of long standing, other acts besides writing are likely to be impaired, especially if they necessitate delicate movements on the part of the affected digits.

Sensory symptoms are often present, and may take the form of a sense of great fatigue, dull pain, tingling, or neuralgia. There is no anæsthesia. The nerves often become tender. The electrical irritability of the nerves and muscles may be normal, or slightly increased or diminished.

Diagnosis.—It is important to make sure that there is

no weakness due to organic disease of the brain, cord or nerves, which is simply revealed by the act of writing.

Prognosis.—If complete rest is given to the affected hand for a long time, the disease may slowly pass away altogether, so that the patient regains the power of writing. If he learns to write with the left hand, the latter may or may not become affected. If he continues to work with the affected hand, the tendency is for the spasm and the sensory phenomena to spread, especially if the general tone of the nervous system is impaired.

Treatment.—The affected hand should have complete rest so far as movements which induce spasm and pain are concerned. The patient should learn to write with the left hand, and to write, moreover, in a correct way from the shoulder. He should use a thick, light penholder. Strychnine and other nerve tonics may be given internally, and local anodynes may be employed over the affected parts. Massage is frequently useful, but must not be applied over tender nerves. One of the first things to be done is to try to remove nerve tenderness, and Poore recommends for this purpose local rest and blistering, with the internal administration of 1-grain doses of potassium iodide combined with arsenic. Poore also recommends rhythmical exercise with galvanisation of the muscles. Care should be taken to rectify any condition of debility that may be present by an abundance of food of a fattening tendency.

xv. Epilepsy

(Falling Sickness. Falling Evil. Morbus Sacer).

Definition.—A chronic disease characterised by recurring attacks of unconsciousness, or of convulsions, or of both these phenomena.

When these attacks cannot be accounted for by organic brain disease, reflex irritation, or morbid blood states, the condition is termed *idiopathic epilepsy*. In some cases, the convulsions involve only a few muscles, or constantly begin in a certain small group of muscles. This is often called *Jacksonian*, or *cortical epilepsy*. It is commonly due to

organic disease (a ' discharging lesion ') causing irritation of, and discharge of energy from, motor centres in the brain. It may be unaccompanied by loss of consciousness. The expression *organic epilepsy* is also applied to that variety which results from a focal lesion in the brain giving rise to local spasm. Epilepsy which is characterised by a brief loss of consciousness without convulsions is called *petit mal* or *minor epilepsy*. When general convulsions are a part of the seizure, it is known as *haut mal*, *grand mal*, or *major epilepsy*. Gowers distinguishes a group of cases intermediate in character between major and minor epilepsy as *medium epilepsy*. In this there is loss of consciousness with slight spasm—either general rigidity, or (in organic cases) local clonic spasm.

Etiology.—The most common time of commencement is the period of puberty. Three-quarters of the cases begin before twenty.

As one epileptic seizure predisposes strongly to subsequent attacks, the question of etiology has to deal specially with the causation of the first attack.

Females suffer rather more than males in the first thirty years of life ; but after thirty, the disease is more likely to show itself in males than in females.

There is epilepsy or insanity in the family in about half the cases. According to Gowers, females inherit the disease especially from the mother, and males especially from the father. Gowers has met with as many as eleven and even fourteen epileptics in one family. According to that writer, other neuroses such as hysteria and neuralgia have no special relationship to epilepsy, and constitutional diseases outside the nervous system are also free from any such relationship. Syphilis is operative only by producing organic brain disease.

In the majority of cases, no exciting cause can be recognised ; but of those which are influential, strong emotion, and particularly fright, in children and young women, and great anxiety in men after middle life deserve special mention.

Injuries to the head, scarlet fever, and reflex irritation

(as by intestinal worms) are occasional causes. Chronic alcoholism is a cause ; the fits generally follow a drinking-bout.

Injury to other parts than the head may be the apparent cause. Thus in one case the fits began after the right forefinger had been crushed in a machine and subsequently amputated. This patient was the subject of elaborate automatic actions, and for some nights before one of these attacks, four years after the injury, he had a sensation of numbness in the injured part.

In a considerable proportion of epileptic women, the fits show a special tendency to occur at the menstrual periods. Many epileptic women remain free from attacks during pregnancy, and it may be lactation also. On the other hand, the disease may begin in pregnancy. Thus a woman began to suffer in her second pregnancy at the age of twenty-five, and suffered in the following pregnancies, from some time after quickening till about a month after delivery. Suckling, except during the first month, and menstruation were quite free from attacks.

Cases which commence in infancy generally arise out of ' teething,' which probably means rickets. These ' teething fits ' or ' dentition convulsions ' may be continued as epilepsy through childhood to puberty and adult life, or there may be an interval of some years between the infantile attacks and those which later on must be regarded as constituting actual epilepsy. Rickets is to be regarded as causing delay, not only in the development of the teeth, but also in that of the higher and controlling motor centres, with the result that lower centres tend to overact. Gowers points out that this relationship to infantile convulsions can be traced in at least a tenth of the cases of epilepsy, and that as rickets is in great measure a preventable disease, a certain proportion of cases of epilepsy might be prevented.

A second and much smaller group of cases beginning in early childhood is distinguished, as Gowers has shown, by these three features : (*a*) unless very severe, the convulsions are confined to one side at first, or throughout ; (*b*) the first attack is often very severe, and indeed there

may be a series of fits going on for hours, and thereafter few or none ; (*c*) the first fit often occurs in an acute illness or after a fall. These fits are due to organic disease at the cortex, and may be associated with hemiplegia (see Infantile Hemiplegia, p. 779).

In yet a third group of cases where the disease can be traced back to early life, the first fits occur within a few days after birth, and are attributable to meningeal hæmorrhage resulting from difficult labour. Such patients are often the subjects of birth palsy and sometimes of mental defect (see Cerebral Diplegia, p. 769).

Epileptic attacks are not often induced immediately by any definite cause, but this occasionally happens. A patient who had been in hospital for some weeks without any attack, took a seizure on the morning of the day on which he was to leave. Some hours afterwards, the house physician playfully asked him what was the meaning of this ; whereupon, instantly, a loud roar initiated a severe convulsive attack. Minor attacks are sometimes induced by strong or otherwise disagreeable impressions of sound, light, smell, etc.

Morbid Anatomy and Pathology.—In idiopathic epilepsy, no characteristic anatomical change is known. The symptoms are referable to a functional (and doubtless often nutritional) disorder of centres in the cerebral cortex, whereby instead of liberating energy under voluntary control, and in a manner regulated by the needs of the body, they set it free suddenly and spontaneously, apart from volition, and often in enormous amount.

Symptoms.—Grand Mal, Haut Mal or Major Epilepsy. —This is the typical falling sickness in which the patient falls to the ground unconscious and convulsed. Consciousness is lost suddenly and completely, so that the patient often hurts himself by striking his face on some hard or sharp object, or he may meet his death by falling into the fire or into water. At the commencement of the attack there is sometimes a cry—the ' epileptic cry.' This may be a loud roar, a scream, or merely a moan, and is probably due to spasm of the larynx. It is not as a rule

heard by the patient. The muscular spasm is at first tonic, so that the body is rigid. The legs are extended. There is often deviation of the head and eyes to one side. The face is at first pale, then flushed and then livid. The tonic spasm is soon replaced by clonic spasm, the jerkings being slight at first and then becoming coarse. There is frothing at the mouth, and the tongue may be bitten at this clonic stage. The froth which issues from the mouth, as well as the pillow, if the patient is in bed, may thus be stained with blood. Then the jerkings of the muscles become less frequent, and after they cease, the patient lies for a time in a condition of coma. Not uncommonly the bladder, and in a few instances the rectum, are evacuated in the attack. This is apparently due to muscular spasm, and not to the unconsciousness. The tonic and clonic stages are both over within two or three minutes, but if the patient is left alone he may sleep for a considerable time after the seizure.

During an attack, the pupils are generally dilated, and the patient is absolutely unconscious. After the attack is over, he may be quite unaware that anything has happened. If his tongue has been hurt, he may attribute the injury to a sharp tooth. He may, however, feel aches all over, particularly in his legs, a consequence, no doubt, of the severe muscular spasm. Wetting of the clothes, and various post-epileptic phenomena such as headache and vomiting, may also indicate to an experienced patient what has happened.

Immediately after a severe convulsion, reflex action is absent for about a minute, and thereafter it is excessive for a time.

Petit Mal or Minor Epilepsy.—Here there are no convulsions, and even consciousness may not be completely lost. There may be simply a brief unconsciousness or giddiness and nothing more. The patient suddenly stops talking, eating or walking, looks strange and pale, and probably drops anything he is holding in his hand. He may resume after a second or two as if nothing had happened, or he may be mentally confused for a time, or he may go through some meaningless automatic action. Urine

is often passed in a minor attack, especially by females, but the tongue is not bitten.

Both major and minor attacks are frequently preceded by a *warning* or *aura*. This often takes the form of a peculiar sensation in the epigastrium which seems to travel upwards to the throat or head, after which consciousness is lost. Special sense auræ are common. The patient sees a flash of light, coloured sparks, etc., or may lose his sight just before he becomes unconscious. He may hear a whistling noise, a sound of bells, etc. Or, again, he may perceive a smell or flavour. Giddiness is a common aura. Psychical auræ also occur, such as sudden horror. A very remarkable and interesting psychical or intellectual aura is that known as the *dreamy mental state*, which is characterised in one of its aspects by the impression that the particular moment of life has been lived before or will be lived again.

In epilepsy due to organic brain disease, and even in idiopathic epilepsy, there may be localised spasm or paræsthesia, for instance in one hand or possibly in the tongue. The occurrence of such localising phenomena, or of special sense auræ, is important as indicating the part of the brain in which the discharge begins.

Post-epileptic Phenomena. — After an attack, the patient may feel quite well, especially if he has slept for some hours after the fit. There may, however, be headache, which is sometimes of great intensity. There may be vomiting, either spontaneously, or after the first food or drink taken. Aching of the muscles has been already alluded to. The exhausting discharge of energy from the motor centres leaves behind it feebleness of the muscles, and in cases where this is more marked on one side, it is sometimes called 'post-epileptic hemiplegia.' In young women, hysterical phenomena may be manifested after an attack. In post-epileptic mania, the patient may be very dangerous to others. Another important automatic action, namely, turning on the face after the fit, involves danger to the patient, since he may be suffocated in his pillow. Other post-epileptic acts are so elaborate as to deserve to be called insane conduct. For instance, a man who was in

the army for three months upset the mess-table on one occasion, turned his comrades out of their beds on another, and at another time, when at drill, charged the officer with fixed bayonet. A young woman in the wards bathed a fellow-patient. A very common act is to undress.

Between the fits, there is frequently observed mental or moral deterioration, which, as Gowers remarks, cannot be ascribed to any one element in the disease. It probably depends largely on congenital instability of the nervous system, though early commencement and long duration of the disease are contributing causes. In many cases, however, the health in the intervals is excellent, and genius of the most brilliant order has over and over again been recognised in association with epilepsy.

Most epileptics take only major attacks. Those who take minor attacks are generally subject to major seizures also. The frequency of epileptic fits varies greatly. A common interval is two or three weeks. The attacks may come singly or in groups. They may occur in the waking state alone, or in the sleeping state alone, or in both conditions.

In rare cases, the *status epilepticus* supervenes. The convulsions follow one another so closely that there is practically no interval of consciousness between them, and this may continue until, after perhaps a day or two, the patient dies with a high temperature. The fits may cease, however, and the patient may then recover, or he may die with delirium and other head symptoms.

Diagnosis.—The severe convulsive attacks witnessed day by day in the streets of our cities are unmistakable, but minor attacks may be misunderstood, and nocturnal epilepsy may long escape detection.

Suddenness in the loss and in the regaining of consciousness, the recurrence of the attacks during years, the absence of a cause of fainting, the passage of urine in the attack, and a liability to convulsive seizures point to epilepsy, and not to *syncope*.

The diagnosis from *hysteria* may be difficult, and it must be remembered that the two sometimes go together (*hystero-epilepsy*). An undoubtedly epileptic fit may be accompanied

or immediately followed by hysterical symptoms. Indeed, the attack may present the features of an ordinary hysterical seizure, including, for instance, the 'crucifixion attitude,' and be distinguishable from simple hysteria by practically nothing except the involuntary evacuation of urine. A sudden onset so that the patient falls and hurts herself, a single cry at the commencement, and biting of the tongue also point to epilepsy. The following table from Gowers may be useful :

	Epilepsy.	*Hysteria.*
Apparent cause.	None.	Emotion.
Warning.	Any, but especially unilateral or epigastric auræ.	Palpitation, malaise, choking, bilateral foot aura.
Onset.	Always sudden.	Often gradual.
Scream.	At onset.	During course.
Convulsion.	Rigidity, followed by 'jerking,' rarely rigidity alone.	Rigidity or 'struggling,' throwing about of limbs or head, arching of back.
Biting.	Tongue.	Lips, hands, or other people and things.
Micturition.	Frequent.	Never.
Defæcation.	Occasional.	Never.
Talking.	Never.	Frequent.
Duration.	A few minutes.	More than ten minutes, often much longer.
Restraint necessary.	To prevent accident.	To control violence.
Termination.	Spontaneous.	Spontaneous or induced (water, etc.).

A patient who complains of waking in the morning with severe headache, sickness, a sore tongue and aching limbs, and of passing his urine in bed, has probably nocturnal epilepsy. A patient who is found lying on his face unconscious has had a convulsion.

When the occurrence of convulsions is recognised, it is important to search for any evidence of organic brain disease (*e.g.*, cerebral tumour, general paralysis of the insane or cerebral syphilis), morbid blood states (*e.g.*, uræmia), or reflex irritation (*e.g.*, digestive disturbance in childhood).

Prognosis.—In a few cases, the disease comes to an end spontaneously, but this is exceptional. It is estimated that about 10 per cent. of cases are permanently cured by treat-

ment. There is but little direct danger to life, for the *status epilepticus* is rare. Patients who turn on the face after an attack are exposed to the risk of suffocation in their pillows. The danger of death by drowning must also be borne in mind.

As regards the prospect of cure, or at least cessation of the attacks, post-hemiplegic epilepsy is unpromising, whilst Jacksonian epilepsy resulting from syphilitic lesions is hopeful. Minor seizures resist treatment more obstinately than major attacks.

In ordinary idiopathic cases, as Gowers points out, an onset after twenty, a short duration, the male sex, a long interval between the attacks, the occurrence only during sleep or only during the waking state, the absence of minor attacks, and the occurrence of an aura are all favourable factors. Curiously enough, an inherited tendency to epilepsy is also favourable. Much depends on perseverance in treatment.[1]

Treatment.—The patient should live a regular and quiet life, with a sufficiency of mental and bodily exercise. A daily bath, followed by thorough rubbing, is to be recommended. Overloading of the stomach and constipation must be avoided. Alcohol should be forbidden unless it is specially indicated.

By far the most valuable medicines are the salts of bromine, and the sodium or the potassium salt should be given every day for at least two years after the last attack ; after this the dose may be gradually reduced. The daily quantity may be given in one dose at night, and this is the best plan if the attacks only occur by night ; or it may be given in divided doses throughout the day, after the principal meals. From 45 to 60 grains may be regarded as the maximum daily supply of the potassium salt for an adult.

[1] As showing that epilepsy is not inconsistent with great attainment or high station, the following may be mentioned as having, according to report, been subject to the disease : Cambyses (conqueror of Egypt), Julius Cæsar, Marlborough, Napoleon, Wellington, Petrarch, Molière, Sheridan, Balzac, Flaubert, and Dostoieffsky. (See Crichton-Browne, ' Dreamy Mental States,' London, 1895, p. 27.)

It is necessary to avoid *bromism*, which is characterised by physical and mental torpor with coldness of the extremities. *Acne*, which is induced in some people by the bromide, is avoided by adding 1 or 2 minims of Fowler's solution to each dose. Digitalis should be added to the bromide in nocturnal epilepsy. Bromide should not be discontinued suddenly, lest the *status epilepticus* supervene.

In cases where bromide fails, as Gowers first showed, borax may be of service if given in doses of from 5 to 15 grains thrice daily after food. Zinc oxide (3 to 5 grains in pill) and belladonna should be tried where bromide fails to arrest minor attacks. Nitroglycerin is occasionally of some service. Phenazone may be added to bromide and borax in obstinate cases. Belladonna, borax, and bromide of camphor are among the drugs that deserve a trial in minor epilepsy.

When a fit sets in, the principal thing is to prevent the patient from being hurt, and to loosen the clothing about the neck. He should be lying on his back, and after the attack he should be allowed to sleep for some time if he is so inclined. If he is in the habit of biting his tongue, a small cork should be placed between the teeth, care being taken that it cannot slip to the back of the mouth.

Sometimes a fit can be arrested. If an aura begins in the hand, a ligature applied higher up the limb may prevent the development of the attack. The inhalation of nitrite of amyl will sometimes also arrest the seizure.

In the *status epilepticus*, chloroform or nitrite of amyl may be administered by inhalation, and chloral hydrate with bromide may be given by the rectum. One of the best remedies, however, is the hypodermic injection of hyoscine hydrobromide. Gowers recommends from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain every four or six hours for a day or two. This writer urges that if morphine is employed, not more than $\frac{1}{12}$ of a grain should be given at one time, lest its effect may combine with the coma of the disease to produce a fatal result.

In Jacksonian epilepsy, where the discharging lesion is not removable by medicine—*e.g.*, by antisyphilitic treatment—surgical intervention may be successful.

xvi. Infantile Convulsions

(Infantile Eclampsia).

The expression infantile eclampsia is only doubtfully applicable to the epilepsy which occasionally begins in childhood. It is not applied to the single fit which sometimes occurs at the onset of an acute fever in a child, and which takes the place of a rigor in an adult; or to the single fit which may result from indigestion. Neither is it used of convulsions which are due to organic disease within the cranium, such as tumour or meningitis. The expression has reference to recurring convulsions of another kind.

Etiology.—By far the most important cause of infantile eclampsia is rickets. As this constitutional disease is associated with late dentition, the fits have long been attributed to teething ('dentition convulsions'). Apart from the powerful predisposing influence of rickets, there may or may not be some exciting cause. Teething may be one, and another is irritation of the alimentary mucous membrane by round worms or by indigestible matter taken in the food. Exhaustion from diarrhœa is another important cause.

Symptoms.—The convulsive attack may come on suddenly, but is often preceded by restlessness; by localised movements such as twitchings of the mouth; or by localised spasm, such as *carpo-pedal spasms* (with inversion of the thumbs and great toes) or *laryngismus stridulus*. As a rule the convulsion is less violent than an ordinary epileptic seizure. The eyes stare or roll about. The body and limbs are stiff, and the head may be retracted. The face is pale at first, but subsequently becomes blue through the interference with respiration. After a varying period, the spasm relaxes, and may or may not give place to clonic spasm. The whole attack may consist of clonic spasm, principally about the face, or the hands and feet.

The fits may be few or many. They may cease within a few days, or recur during many months.

Diagnosis.—Organic disease within the head must be excluded by noting the accompanying symptoms, which may point to tumour, meningitis, infantile hemiplegia, etc.

Unilateral fits suggest intracranial disease. Convulsions beginning immediately after birth indicate meningeal hæmorrhage. The possible influence of acute disease; of mechanical congestion of the brain, as in whooping-cough; and of an indigestible meal or intestinal worms, must be borne in mind. In the absence of these conditions, there is likely to be some general cause such as rickets or exhaustion. Rickets is recognised by its special symptoms, and by the changes in the skull and in the ends of the long bones. Exhaustion is often due to diarrhœa, and the latter may in addition give rise to hydrocephaloid, in which pallor, stupor, contraction of the pupils, and depression of the anterior fontanelle are present.

Prognosis.—Most cases recover, but if the fits are frequent and severe, death may take place from exhaustion. In a few cases, epilepsy will supervene later on.

Treatment.—The cause should be searched for, and, if possible, removed. For severe convulsions, chloroform inhalations should be given at once; or 20 grains of sodium bromide may be administered in one dose by the rectum, even to a young baby. The bromide should be continued in smaller doses for some days (3 to 5 grains thrice daily for a child under one year). If the *status epilepticus* develops and persists in a severe form, it may be necessary to resort cautiously to hyoscine hydrobromide ($\frac{1}{200}$ to $\frac{1}{100}$ grain hypodermically). The warm bath and the lancing of swollen gums are old-established remedies, but their value is doubtful. Rickety children should get cod-liver oil and chemical food; and their diet, clothing and hygienic conditions must have careful attention.

xvii. Neuralgia.

Definition.—Pain in a nerve not accounted for by disease in the nerve or nerve centre.

In practice the term is also used to include pain at a distance from its cause, and pain which persists after removal of its cause.

Etiology.—Children fortunately are not liable to neuralgia, but the disease is common about puberty and adolescence.

It is predisposed to by an inherited tendency to neuroses. It is favoured by anæmia, and this has doubtless much to do with the frequency of the ailment in girls at the age when chlorosis is common. Neuralgia is also favoured by debility of any kind, such as may be induced by lactation, menorrhagia, influenza, overwork and insufficiency of sleep. Among other causes are morbid blood states resulting from lead poisoning, gout, rheumatism, diabetes, malaria, etc.

The most important exciting cause is exposure.

Neuralgia may be due to irritation of a nerve, as in dental caries, or to neuritis.

Morbid Anatomy and Pathology.—In pure neuralgia, no constant anatomical change is known. The pain is probably due to overaction of sensory centres in the cerebrospinal axis.

Symptoms.—The pain varies much in character and intensity. It is generally unilateral, and often persists in one part for a long time. It is usually paroxysmal, and is frequently absent altogether during a certain portion of the twenty-four hours. Thus it may set in every day after dinner, or whenever the patient becomes warm in bed. While it is present, the pain may be a continuous gnawing, aching or boring ; or it may come at intervals of seconds or minutes in the form of momentary, darting, stabbing or lightning-like twinges of intense severity. When very severe, the pain may radiate to other parts. The pain may be influenced by temperature, and may be relieved by firm pressure.

Tender points can frequently be discovered in the course of the affected nerve (the ' tender points of Valleix '), and these are specially apt to exist where it passes through a foramen, or through a fascia, or where it divides. Slight numbness and diminution of tactile sensation may be detected in connection with neuralgic attacks. The vasomotor system may be deranged, as shown by pallor or flushing of the skin, throbbing of the vessels and even œdema. Disturbance of the glandular functions may be manifested by local sweating. Trophic changes may occur in the form of loss or greyness of the hair. Twitchings of

the facial muscles may occur in cases of trigeminal neuralgia.

Varieties.—*Trigeminal neuralgia* (*Trifacial neuralgia, Tic douloureux, Prosopalgia*) is one of the most common forms of the disease. It may involve one, two, or, less commonly, all of the divisions of the fifth nerve.

When the *first* or *ophthalmic division* of the nerve is involved, the seat of pain is the forehead, upper eyelid, and side of the nose. The expression ' brow-ague ' is still applied to neuralgia of the ophthalmic division, although in this country, at the present day, malaria is rarely its cause.

Tender points may be found at the supraorbital notch, in the upper eyelid, at the inner angle of the orbit, at the side of the nose where the superficial branch of the nasal nerve emerges between the nasal bone and the upper lateral cartilage of the nose, or in the eyeball itself.[1]

In neuralgia of the *second* or *superior maxillary division*, the pain is referred to the cheek, jaw, ala of the nose, or some more limited portion of the territory of the nerve. It may be referred to one or more of the teeth, even though these are healthy. In one case, caries of the second left molar in the lower jaw caused severe intermittent neuralgia in the healthy first upper molar of the same side. The pain in the upper tooth ceased as soon as the diseased lower tooth was extracted.

[1] Frontal headache is caused in some people by the eating of ices. Thus, in one instance, a portion of ice was swallowed rather quickly, and the cold mass caused severe pain in the stomach for some seconds ; there was simultaneous severe pain at the root of the nose on the right side only, particularly in the angle between the nose and the supraorbital margin. In another case, the eating of ices always caused severe pain in the forehead, lasting for about half an hour, and not associated with pain in the stomach. The sufferer, who was a medical man, stated that nothing but ices produced such pain.

As another instance of the modes in which frontal neuralgia is induced may be mentioned the case of a woman who was treated for dyspepsia with an acid mixture. After she began to take this, troublesome pain set in at the lower part of the forehead, over the root of the nose. An alkali was therefore substituted for the acid, and the pain went away.

Tender points occur at the place of emergence of the infraorbital nerve, over the malar bone, on the gum below the malar bone, and at the side of the nose.

In neuralgia of the *third* or *inferior maxillary division*, the pain may involve the lower jaw, tongue, ear, and temple. Intense pain may be restricted to the territory of the lingual nerve.

Tender points may be detected at the mental foramen, in the temple, just above the zygoma in front of the ear, and at the tip of the tongue.

Trigeminal neuralgia is sometimes aggravated by cold, by movements of the jaw in mastication, and by pressure. Reflex spasm of the facial muscles may accompany severe attacks, and vasomotor, trophic and other disturbances are observed in some cases.

Cervico-occipital neuralgia involves the part of the neck supplied by the first four cervical nerves, and the back of the head as far as supplied by the great occipital (a branch of the second cervical). This pain is usually continuous and not very intense. It is frequently bilateral, and may be confined to the posterior branches.

An important tender point is situated midway between the mastoid process and the middle line, where the great occipital nerve emerges. The scalp may be extremely sensitive.

Cervico-brachial neuralgia involves the territory of the four lowest cervical and first dorsal nerves. It is usually continuous, with paroxysmal exacerbations. It is aggravated by movement, and may radiate to the side of the chest and thus simulate angina pectoris.

Tender points are found in the axilla, at the posterior border of the deltoid muscle, over the musculo-spiral nerve 3 inches above the outer condyle, over the ulnar nerve in the groove between the olecranon and inner condyle, at the ulnar side of the annular ligament, and where the radial nerve becomes superficial.

Dorso-intercostal and *lumbo-abdominal neuralgias* correspond to the territories of the upper and lower intercostal nerves. Among the former may be included the inframammary pain of anæmic women. It is most common on

the left side, and is distinguished by being continuous, and not much if at all influenced by a deep inspiration.

Tender points in these neuralgias are found over the places where the principal branches of the intercostal nerves end, viz., near the spine, near the middle line in front, and in the lateral region. In lumbo-abdominal neuralgia, a tender point may be present in the scrotum or labium.

Herpes zoster is often associated with and followed by severe pain in the territory of an intercostal nerve.

Coccygodynia, or neuralgia in the region of the coccyx, is most common in women.

Reflex or *sympathetic neuralgia* is pain felt in a part other than that in which its cause exists. Thus pain in the knee may be caused by hip-joint disease. And intermittent neuralgia in the second division of the fifth may be caused, as in the case mentioned on p. 838, by painless caries in a lower molar tooth, which is supplied by the third division. A partial explanation of such cases is that when the impulses set up in the nerves of the diseased parts reach the grey matter of the spinal cord, bulb, or pons, as the case may be, they diffuse themselves in that grey matter, and thus influence neighbouring centres which are connected with the reflexly painful parts.

A very important and extensive group of these reflex or reflected neuralgias is constituted by the *visceral neuralgias*. These are superficial pains induced by visceral disease—*e.g.*, the pain in the shoulder caused by hepatic abscess, the pain at the back of the chest in gastric disease, and the pain in the thigh in renal colic.

Epileptiform neuralgia (*neuralgia quinti major*) is a terrible form which involves the fifth nerve in whole or in part. The attacks are of sudden onset, of agonising intensity, and of short duration. They may occur once or many times in a day, and may be accompanied by spasm of the facial muscles. The patient has generally reached or passed middle life.

Diagnosis.—This depends on the absence of any discoverable organic cause, the absence of signs of damage to nerve fibres—*e.g.*, muscular wasting, or marked and persistent anæsthesia—the frequently unilateral distribution of the

pain, the correspondence in distribution with some nerve, the absence of early and marked tenderness of that nerve, and the paroxysmal nature of the affection. It is important not to mistake for mere neuralgia the bilateral pains of tabes, the pains occurring in diseases of the spine and spinal membranes, or the pains of deep-seated aneurysm.

Prognosis.—This is favourable, if a removable cause, such as anæmia, exists. It is not so favourable if the disease has lasted long. After middle life, neuralgia, and especially epileptiform neuralgia, is apt to be extremely obstinate. Neuralgia is not dangerous to life, but is liable to recur.

Treatment.—Any cause which operates by producing anæmia or constitutional debility or a morbid blood state must be removed if possible. Under this heading will come the treatment of chlorosis, leucorrhœa, dyspepsia, gout, malaria, etc. Local causes which more directly influence the part which is the seat of pain must also receive attention. This will include the treatment of dental caries, etc. As a rule the two kinds of causes must be borne in mind : the depressed constitutional state, and the local irritation which determines the actual seat of pain. Pain, as Romberg taught, is the prayer of a nerve for healthy blood.

Tincture of the perchloride of iron (10 to 20 drops) with sulphate of quinine (1 to 3 grains) three times a day constitutes one of the most valuable of all remedies. The daily intermittent form will sometimes be permanently arrested by one or two doses of quinine. Rest and a change of air are desirable. Among other remedies are arsenic, strychnine, phosphorus, the hypophosphites, tincture of gelsemium, phenacetin, phenazone, and butyl chloral. Nitroglycerin may be tried in combination with strychnine and cannabis indica or gelsemium. Morphine or cocaine may be given hypodermically in urgent cases, but only by the physician personally. Cimicifuga is recommended in rheumatic and gouty cases.

Among local measures are anodyne and irritant liniments, ointments, and plasters ; the actual cautery ; and electricity. A favourite preparation is a mixture of equal parts of the liniments of aconite, belladonna and chloro-

form, to be rubbed into the painful part. Or one part of each of the first two liniments is added to two parts of glycerin, and the mixture is employed to saturate several thicknesses of lint, which is then placed on the seat of pain, covered with oiled silk, and left in place for hours if need be. Methyl salicylate may also be employed as a wet dressing. In some instances, a small blister is advisable. Electricity may be used as a counter-irritant in the form of a painful faradic current; or as a sedative in the form of the continuous current with the positive pole at the seat of pain. In obstinate cases, the affected nerve may be stretched or resected. In epileptiform neuralgia and other varieties which are associated with exquisite agony, and make the sufferer's life a burden to him, the Gasserian ganglion may be removed in whole or in part, or ganglia of the posterior roots may be excised, according to the seat of pain. Horsley states that the death-rate after excision of the Gasserian ganglion is only 7 per cent. The mortality is practically confined to individuals who have reached the age of fifty-five, and is mostly due to hæmorrhage resulting from arterio-sclerosis, which is very constantly associated with disease of the ganglion.

xviii. Migraine

(Megrim. Hemicrania. Paroxysmal, Sick, Nervous, Bilious, or Blind Headache).

Etiology.—Migraine is often inherited; Gowers states that inheritance can be traced in more than half the cases. Several members of a family are often attacked. Females suffer more frequently than males. A third of the cases begin, according to Gowers, between five and ten years of age. Puberty and early adult life are the two other most common periods of onset. Gout probably favours its development. The disease may be originally caused, and paroxysms may be induced, by mental or bodily fatigue, loss of sleep, anxiety, excitement, a hot atmosphere, or any condition which temporarily impairs the nervous tone. When it begins in early life, there is commonly no

recognisable cause. The attacks are probably reflex in some cases—*e.g.*, from uterine disorders, menstruation, dental caries, refractive errors, strain of the eyes, adenoids in the naso-pharynx, or digestive disturbance. Writers comment on the large number of individuals distinguished by their intellectual ability who have been victims of migraine.

Morbid Anatomy and Pathology.—No anatomical changes are known to account for the phenomena of this disease. Its pathology is a matter of speculation. It used to be supposed that the attack depended upon disturbance of the stomach or liver, but this view is not now held to any extent by scientific observers. Migraine has also been regarded as a form of neuralgia, though the occurrence of vomiting, the long intermissions, the continuance of the disease during many years, and the ineffectiveness of treatment differentiate it from the ordinary forms of neuralgia. Two principal theories of migraine are held at the present day. Latham holds that the disease is referable to the sympathetic system. Thus, when fatigue and other depressing influences lower the tone and controlling power of the cerebro-spinal system, the uncontrolled sympathetic system is apt to cause contraction of bloodvessels, and so produce the sensory disorders which precede the headache. This excitement of the sympathetic is followed by exhaustion of that nerve with consequent dilatation of bloodvessels and headache. As the state of the vessels varies in different cases, many writers hold that there are two varieties of the disease, spastic and paralytic.

The state of the vessels, however, may vary in different attacks in the same person, even though no difference in the pain can be recognised. For instance, Lauder Brunton, one of the many distinguished scientists who have been able to describe the disease from personal experience, says that in his case the carotid is always dilated, and feels as thick as the thumb ; whereas the temporal artery is sometimes dilated and pulsating furiously, and on other occasions contracted and hard like piano wire.[1] Other important difficulties exist in the way of accepting the vasomotor theory of migraine.

[1] 'On Disorders of Assimilation, Digestion,' etc., 1901, pp. 250, 251

According to the other theory, which is due to Edward Liveing, the paroxysm of migraine is a 'nerve-storm,' characterised by a temporary disturbance of function in cerebral nerve centres. The vasomotor disturbance, on this theory, is secondary. If this view be accepted, the paroxysm of migraine may be regarded as a sensory analogue of the epileptic convulsion. Gowers has called attention to the resemblance between these two paroxysmal neuroses in the ratio between the sexes, and in the large number of cases which commence at puberty.

Symptoms.—Headache is the most constant and characteristic symptom, but in a typical attack there are two stages. In the first or premonitory stage there is some sensory disturbance—in most cases, a disorder of vision, such as spectra or hemianopia. After fifteen or thirty minutes, the sensory disturbance subsides, and the second stage, characterised by headache and other symptoms, thereupon sets in. The attack commonly ends after some hours with nausea and vomiting. In some individuals, however, there is only the first stage, so that they escape without headache. In others, again, the headache seems to be the first symptom, so that the premonitory stage is either absent or so slightly marked as only to be detected by careful investigation.

1st Stage.—Visual disturbance is often the first symptom, but is only met with in about half the cases. The loss of sight is seldom complete. When it is unilateral, it takes the form of hemianopia, the corresponding halves of both fields being involved. In some cases a dim spot appears in the centre of the field, and grows in size, sometimes clearing in the centre as it increases in diameter. Or the dimness may commence in the lateral part of the field, and though it spreads, it does not usually pass beyond the middle line, so that here again it produces a hemianopia. In the area of dimness of vision there is commonly an appearance of glimmering or rapid tremulous movement. In some cases, a bright spot is the first change, and as this grows it may become dim in the centre. The outer boundary of the abnormal visual phenomenon is often made up of zigzag

lines which have given rise to the name 'fortification spectrum' (*teichopsia*). These lines may be associated with colours. The visual disturbances just described always involve both fields.

In some cases the visual disturbance may be very trifling and consist merely of flashes of light, sparks, or simple hemianopia without spectra. In rare cases, symptoms have been noted in connection with the auditory and olfactory mechanisms comparable to the common visual disturbances, and including therefore evidences both of irritation and of loss of function.

In a few cases, corresponding phenomena occur in the limbs, and chiefly in the arm of one side. There is tingling with numbness or even anæsthesia. The patient feels that the part is 'asleep.' Occasionally the sensory disturbance is accompanied by slight impairment of power. The disturbance may pass from the limb to the lips and tongue. This group of symptoms may take the place of visual phenomena as the first stage of the attack, but as a rule they set in as the visual disturbance is passing off. Their ordinary duration is ten or fifteen minutes.

Aphasia is another occasional disturbance, and is usually, as might be anticipated, of the sensory type. If hemianopia is present in such a case, it is generally right-sided; and if the sensory disturbance involves the limbs, it is generally the right arm that suffers. These phenomena, of course, point to a disturbance of the left cerebral hemisphere in a right-handed individual.

In other cases, there are no ocular symptoms, but the patient is depressed, restless, and uneasy, and the extremities may be cold. This stage may continue for half an hour or so before the headache begins; or it may last for a number of hours, so that the patient goes to bed in this state and wakens early with the severe headache. Mental confusion and loss of memory may also occur in the first stage, either early or late.

2nd Stage.—The headache is usually severe, and is often, though by no means always, unilateral. Unilateral pain is generally on the opposite side from sensory symptoms

connected with the eyes and limbs. The pain frequently has a boring character, and increases in severity as the ocular disturbance subsides. It may begin in a very limited area of the temple or forehead, and from this it gradually spreads over a great part of the head, on the same side, or on both sides. The eyeballs ache and are tender, but tenderness of the head is unusual. The patient chooses the recumbent posture, as his suffering is apt to be worse if he sits or stands. Movement, noises, and bright light aggravate the pain.

As the headache is increasing, or after it has reached its maximum, nausea sets in. Not only is there complete disinclination for food, but food that is swallowed remains undigested and unabsorbed, and may be vomited many hours later in the same condition as when it was taken. When the headache is at its worst, or when it is abating, retching and vomiting often occur, and if the headache is already past its worst, vomiting may bring the attack to an end.

The face may be pale throughout an attack, or it may be pale at first and afterwards flushed when the headache becomes severe. The superficial arteries of the head may be contracted at first and dilated later on; and, as has been already mentioned, the carotid may be dilated whilst the more distal temporal artery is strongly contracted. Perspiration and slowness of the pulse are occasionally noted.

After continuing for a number of hours, often for a whole daytime, and occasionally for several days, the headache subsides, and the patient falls asleep. The sleep may be short and troubled, but the sufferer awakes free from pain, though listless and worn out.

The attacks may recur during a period of many years. The interval between the paroxysms varies, but is often a few weeks. In women the attack frequently occurs at the close of the menstrual period.

Optic atrophy and migraine are occasionally met with in combination—probably a mere coincidence.

Diagnosis.—The paroxysmal character of the headache, its recurrence at intervals of weeks or months during an

indefinite number of years, the associated sensory disturbances, and the nausea and vomiting, taken in conjunction with the unimpaired general health, are quite distinctive of migraine. The sensory disturbances may occasionally suggest *petit mal*, but an epileptic aura is a very transient phenomenon.

Prognosis.—There is no danger to life. After middle life, both in men and in women, the attacks often cease.

Treatment.—The patient must avoid what he knows to be causes of an attack—*e.g.*, excitement, crowded rooms, irregularity in meals, constipation, and fatigue of body or mind. Errors of refraction and nasal adenoids must be attended to. Gowers strongly recommends the regular administration of nitroglycerin in the intervals, just as bromide is given in epilepsy. It may be combined with strychnine and an acid. Iron, potassium bromide or sodium salicylate may be of service in some cases.

In the paroxysm, complete rest in a quiet and darkened room is the best treatment. If the patient feels chilly, hot bottles should be placed at his feet. The drug given in the interval should be stopped. Analgesics, such as phenacetin or phenazone, may be tried for the pain ; and strong tea or coffee, or 15 grains of guarana powder, may be given. Menthol may be applied over the seat of pain, and a mustard poultice or plaster over the back of the neck. A hot mustard foot-bath may be helpful.

Little good, on the whole, need be expected from remedies swallowed after the paroxysm has fairly begun, since gastric digestion is at a standstill. If, however, the patient can tell of an approaching attack some hours beforehand, as in the case of women in whom it bears a constant relation to the menstrual period, the prompt administration of phenacetin may prove highly beneficial.

xix. Hysteria.[1]

Etiology.—The disease is many times more common in women than in men. It is also much more frequent in

[1] From Greek ὑστέρα = womb, the disease having been formerly regarded as a result of uterine disorder.

boys than in adult males. Half of the female cases begin in the second decade of life, and more than a quarter in the third decade. Very few cases begin before the sixth year. The disease begins before puberty in a larger proportion of males who suffer from it than of females. Hereditary tendency is an important predisposing cause ; in the parents and other relatives there may be either hysteria itself or some other neurosis such as insanity. The disease is almost confined to the civilised races, and the latter are not all alike liable. Thus the most severe forms are seen chiefly in France. Hysteria is also favoured by prolonged anxiety with deficient sleep, as in nursing a sick relative ; and by luxurious habits, alcoholism, sexual excesses, enforced celibacy and unhappy circumstances in life. When it is present, it is apt to be aggravated at the menstrual periods. Syphilis in prostitutes is sometimes associated with severe hysteria.

Among the causes which may determine the outbreak of the disease are emotion, moral contagion, debility, painful impressions, shock to the nervous system, injury or disease of any part of the body, and especially disease of the nervous system. Imitation or moral contagion is seen when a hysterical paroxysm in a hospital patient is followed by similar attacks in other patients in the same ward. When the disease results from a local lesion, the hysterical symptoms may be localised in the seat of lesion, as in the larynx or knee-joint. Hysterical phenomena may be witnessed in connection with such nervous diseases as insular sclerosis, cerebral tumour, meningitis and epilepsy.

Pathology.—The patient is not merely a hypochondriac who fancies she is ill when she is not ; or a malingerer who pretends that she is ill when she is not ; although the laity are very apt to put one or other of these interpretations upon hysteria and its symptoms. Hysteria is a real disease and is to be regarded as a functional disorder of the nervous system. The highest nervous centres are primarily at fault, but it frequently happens that centres of a lower grade are deranged secondarily. Convulsions and certain other manifestations of hysteria are attributable to loss of

that control over the lower and more automatic centres which is normally exercised by the high centres associated with volition. Paralysis is accounted for by failure of the volitional centres to initiate motor impulses.

Symptoms.—These are both continuous and paroxysmal, but no distinct line of demarcation can be drawn between the two groups, since certain phenomena may be paroxysmal in some patients and persistent in others. The continuous symptoms depend to a large extent on an abnormal mental and emotional condition. The paroxysmal symptoms include motor, sensory and other phenomena.

(1) *Mental Symptoms.*—The patient may be unduly emotional, and excited too readily to laughter or tears. Moreover, the emotion may be perverted, so that laughter is induced where tears would be more appropriate, and conversely. Or a hysterical fit may take the place of the usual manifestations of emotion. Moreover, the power of concentrated and sustained attention, which is one of the very highest of the intellectual faculties, is reduced. Will-power, judgment, and self-control are deficient. The patient is apt to be irritable and oversensitive, and to be the victim of self-consciousness.

In some cases, the moral nature is impaired, and the patient practises deception. This is doubtless often due to, or developed by, the ill-directed kindness of friends who respond too readily to the morbid craving for sympathy which is one of the phenomena of the disease. Fostered in such ways, the craving grows, and uncontrolled by healthy moral fibre or will-power, it invents new and often dishonest means of attracting the attention and sympathy of others.

Hallucinations are occasionally present in hysteria, and are likewise due to involvement of centres of a high order. The patient may become definitely insane—maniacal, melancholic, or demented.

Trance or *lethargy* is chiefly seen in hysterical subjects, though it is not confined to such. It is practically identical with the hypnotic state, but it comes on spontaneously

instead of being, like ordinary hypnotism, induced by an operator. It may follow a hysterical fit, or may be a result of mental or bodily exhaustion. It usually sets in suddenly, and lasts for hours, days, weeks or months. It is characterised by marked pallor, relaxation of the muscles, loss of reflex action and complete unconsciousness. Sometimes, however, the reflexes are preserved or even excessive, and occasionally the patient is conscious of what is going on around, though completely unable to indicate that such is the case. The cardiac action is very feeble, and the radial pulse may be imperceptible. Breathing may also be extremely feeble. In the most profound trance ('death-trance'), the patient appears to be dead, even to a careful observer, and in this condition she may be buried. The attack ends gradually or suddenly, and may recur. In prolonged attacks, remissions usually occur, in which a little food may be taken.

Catalepsy is a variety of trance in which there is *plastic rigidity* of the limbs (*flexibilitas cerea*). It is usually either associated with hysteria, or induced by a recognised cause of hysteria, such as strong emotion, nervous exhaustion, etc. The attack is often sudden and lasts for minutes, hours or days. In many cases it recurs. At first the muscles are rigid, so that passive movement is resisted, but after a time the rigidity diminishes. The limbs may then be moulded to any posture, and they retain this posture as if they were made of wax. They gradually, however, yield to the influence of gravity. Consciousness is impaired in greater or less degree.

(2) *Motor Symptoms.* — These include (*a*) tonic and (*b*) clonic spasm, (*c*) paralysis, and (*d*) convulsions.

(*a*) *Tonic spasm* may involve one or more muscles. *Hysterical contracture* is a form of tonic spasm which is very persistent, and may not pass off for months or years. It usually sets in after a hysterical fit or a local injury, or in connection with local pain. It may involve one or more of the limbs. The arm is most often affected, the elbow and wrist being rigidly flexed. The leg, on the other hand, if the seat of contracture, is rigidly extended with the toes

pointing downwards. The spasm is sometimes hemiplegic or paraplegic in distribution; or all the limbs may be involved.

Similar spasm of the masseter muscles constitutes *trismus*. The features which distinguish this condition from tetanus are mentioned at p. 165.

Tonic spasm may involve a portion of a muscle and give rise to the appearance of a tumour. Localised spasm in certain abdominal muscles is probably concerned in the production of the *phantom tumour* of the abdomen.

Severe contractures generally persist in sleep, but may relax when the patient is deeply under the influence of chloroform. Even after lasting for years they may cease suddenly under the influence of strong emotion. Faradisation kept up for several minutes, so as to exhaust the muscle, may cause relaxation, and may thus be useful both in diagnosis and in treatment.

(*b*) *Clonic spasm* is common in the form of *tremor*, especially under the influence of excitement, attention and movement. Clonic spasm occasionally involves the muscles of the neck, causing the head to move backwards and forwards.

(*c*) *Paralysis* is frequent in hysteria, and assumes different forms. The most common variety is that which causes *hysterical aphonia*, viz., paralysis of the laryngeal adductors. The palsy is incomplete, since the cords, though not brought together in phonation, are yet adducted in coughing. Hysterical aphonia may be the sequel of a laryngeal catarrh, or may be brought on by emotion. It may pass off suddenly, but is apt to recur. In rare instances, there is paralysis of the abductors with alarming symptoms, but with preservation of the voice.

Mutism and *stuttering* are also met with. Hysterical mutes usually retain the power of reading and writing.

Paraplegia is another hysterical palsy, and is often due to emotion. It often sets in suddenly and increases in degree, but does not become complete. The patient may be unable to walk, or may be able to move about with difficulty. The nutrition and electrical irritability of the muscles are usually unimpaired, the patient retains control over the sphincters, and there are no bedsores. The deep reflexes may be normal

or slightly increased. There may or may not be loss of sensation. The phenomena of *astasia-abasia* (*q.v.*) are occasionally observed.

Hysterical *hemiplegia* may set in gradually or suddenly, and is usually accompanied by hemianæsthesia. As Gowers points out, it differs from ordinary organic hemiplegia in certain respects : the paralysis is never complete ; the leg is often more affected than the arm ; the face usually escapes entirely ; and cutaneous reflex action is not lessened on the paralysed side.

Hysterical *ptosis* sometimes occurs on one or both sides.

Various forms of *ataxy* or inco-ordination of movement are met with in hysterical subjects, either alone or in association with paralysis.

(*d*) *Convulsive attacks* or *hysterical fits* are familiar incidents in connection with hysteria, and may result from emotional disturbance. The fit is often preceded by laughing, crying, the *globus hystericus*, headache, giddiness or some other symptom. The onset is not absolutely sudden as in epilepsy, and the patient in falling does not hurt herself. She may give utterance to a choking cry in the course of the attack, but not to the moan or shriek which may usher in an epileptic seizure. The limbs and trunk may be rigidly fixed, the trunk in *opisthotonos*, and the upper limbs in abduction, so that the patient assumes the *cruciform attitude*. Rigidity, however, may alternate with wild irregular movements of the limbs and head. The patient has the aspect of being conscious in a way that is not seen in ordinary epileptic seizures. She does not bite her tongue, though she may bite her lips and hands, and may bite other people and things. She may talk in the course of the fit and even make reference to an individual who is present, but consciousness is probably often impaired, and after a severe attack, the patient is usually unaware of its occurrence. The convulsion is frequently much more protracted than an epileptic attack. It may end with a fit of weeping, and with the passage of a large quantity of pale urine of low specific gravity. In some instances, the convulsive seizures recur at short intervals during many hours, so that the question of the *status*

epilepticus arises. The latter condition, however, is associated with a marked elevation of temperature ; whereas, as Charcot pointed out, there is but a slight rise in the case of the corresponding hysterical condition (*status hystericus*). There is no incontinence in pure hysteria, but an epileptic seizure may be the exciting cause of hysterical symptoms.

The expressions 'hysteroid convulsions' and, in France, 'hysteria major' and 'hystero-epilepsy' are applied to severe hysterical seizures which are contrasted with the 'hysteria minor' of French observers. *Hysteria minor* represents the slightest form of convulsive attacks in which consciousness is not lost. In *hysteria major*, as observed by Richer and others in French patients, the rigidity, movements and mental disturbance are specially well marked. The attack may be preceded by a prodromal period of hallucinations. The fit itself presents first an epileptic stage, characterised by sudden loss of consciousness, followed by tonic spasm and then by clonic spasm ; secondly a stage characterised by opisthotonos and violent movements, and thirdly a stage of great mental and emotional disturbance. Pressure over the ovary will induce or arrest an attack as the case may be.

(3) *Sensory Symptoms.*—These include both exaggeration and diminution or loss of the different kinds of sensibility, either separately or together. Thus there may be *hyperæsthesia* with *intolerance of light*, *tinnitus aurium*, etc. *Neuralgic pain* and *tenderness* are common in various situations, such as the mamma, the left inframammary region (as in anæmia) and the cervical and dorsal spines. The *clavus hystericus* is a very severe form of headache which is so called because the patient feels as if a nail were being driven into the top of her head (*clavus* = nail). Sometimes a *joint*, and especially the knee or hip, becomes the seat of pain, possibly after a slight injury. The patient complains of constant and intense pain in it, and cannot bend it, or allow it to be passively flexed. The nature of the condition is recognised by the absence of any signs of inflammation after a couple of days, and by the perfect mobility of the part when the patient is placed under the influence of

chloroform ; complete and immediate cure may follow the anæsthesia and passive movement. Another important seat of tenderness is situated internally to the anterior superior iliac spine, and has been supposed to correspond to a tender ovary. Pressure on this part in such patients may give rise to considerable distress or actual pain, with faintness, the *globus hystericus*, and even a convulsive paroxysm. When compression of any spot or region induces a hysterical seizure, such a part is described as a *hysterogenic spot* or *zone*.

Paræsthesiæ, such as tingling and numbness, are common, and are often unilateral.

Diminution or loss of sensation is also common, and frequently takes the form of *hemianæsthesia*. The loss may be complete, extending up to the middle line of the skin and mucous membranes, and including the *special senses*. The visual loss takes the form of *crossed amblyopia*, in which visual acuteness and the visual field are both greatly reduced on the anæsthetic side and much less affected on the other side.[1] Contraction of the field is often more marked for blue than for red.

When the loss of sensation is incomplete, it may be ' dissociated,' certain forms of sensation being preserved and others lost. Thus there may be complete hemianalgesia, with mere blunting of tactile sensibility. The *plantar reflex* is often lost in hysteria, even though common sensation in the feet be preserved. If it is present, it is flexor in type. The *knee-jerks* are exaggerated.

Hemianæsthesia may set in spontaneously, or after a fit or strong emotion. It may be a transient phenomenon, and may shift from side to side, either of its own accord, or on the application of metals, magnets, blisters, etc. The fact that such a ' transfer ' can be effected by artificial means was discovered by Charcot. Before many hours have elapsed, however, the anæsthesia generally transfers itself back to the side originally involved.

[1] In hemiplegia with hemianæsthesia and loss of the special senses resulting from a lesion of the internal capsule, the visual defect is lateral hemianopia.

Anæsthesia is occasionally present in the legs along with paralysis. It may also be distributed in irregular patches over the trunk and limbs, though the patient is not always aware of its presence. When it involves the distal segment of a limb, with a well-defined boundary between the normal and anæsthetic portions, it gives rise to the 'glove' or 'stocking' anæsthesia.

(4) *Visceral and Vasomotor Symptoms.*—Disturbances of the circulatory apparatus are met with in the form of *palpitation, tachycardia, faintness, flushing of the face,* etc.

Involvement of the respiratory and laryngeal nervous mechanisms may give rise to coughing, or to sounds which are obviously imitative of the cries of animals or other familiar noises. The *barking cough of puberty* (*cynobex*), an hysterical symptom sometimes noted in boys, is often due to masturbation. Among other respiratory symptoms there may be very *rapid breathing*, which is sometimes wrongly called 'hysterical dyspnœa.' Hiccough may also be mentioned here.

The *globus hystericus* is a well-known symptom in which the patient feels as if a lump travelled up the gullet to the throat, where it gives rise to a feeling of choking. In severe cases, there is probably actual spasm of the pharynx. *Dyspepsia* of all kinds, *anorexia, dysphagia, œsophagismus, vomiting* and *constipation* may be present.

Anorexia nervosa (*anorexia hysterica, apepsia hysterica, atrophia nervosa*) is characterised by absolute loss of appetite, with constipation, restlessness, amenorrhœa, and ultimately extreme emaciation. Death occasionally ensues, and the post-mortem examination throws no light on the symptoms. If, however, the nature of the case is recognised, and the proper treatment—*e.g.*, by Weir Mitchell's method—is enforced, recovery is likely to take place. Now and then, the patient fails to recover, and yet survives for many years. Anorexia nervosa furnishes the cases of 'fasting girls,' but there is no doubt that in many of them deception is practised in the matter of food.

Retention of urine, ischuria, and even complete *anuria* lasting for days may be met with. The *temperature* may be

slightly elevated in severe cases. In some instances, by compression or friction of the bulb of the thermometer, or by some less explicable means, the patient is able to record a temperature which, before the nature of the case has been investigated, may appear truly alarming. Thus 108·4° was noted in the case of a hospital nurse who was under my care some time ago.

Diagnosis.—It is important to guard against diagnosing as hysterical a case in which structural or other serious disease is present. The first thing to do is to search carefully for any organic cause, bearing in mind that disease of the nervous system, functional or organic, is a common cause of hysteria. If every such explanation can be set aside, the age and sex of the patient, the previous history of the case, the apparent causation by emotion, and the special symptoms present, must be taken into account. In the case of certain symptoms, such as joint affections, contractures, and phantom tumours, general anæsthesia may be desirable to allow of a thorough investigation. Insular sclerosis and epilepsy must be specially borne in mind in cases which present hysterical symptoms (see pp. 785 and 831).

Prognosis.—Hysteria rarely causes death, though this may result from persistent vomiting, anorexia, or the rare form of laryngeal spasm characterised by paralysis of the abductors. Many of the symptoms may be removed, but the underlying tendency is likely to persist and allow of recurrence, or of new manifestations. Insanity may supervene. Nevertheless, if more favourable conditions of life arise, and the patient becomes fully occupied with, and interested in, work and hobbies of a healthy character, even the morbid underlying tendency may cease to exist.

Treatment.—Any possible cause of the disease, whether local or general, should be removed. The patient should lead a quiet regular life, in the country if possible ; and it would be well if she could adopt a hobby, in addition to having an agreeable regular occupation. She should have a daily tepid or cold bath or douche. Tonics should be given, and the bowels must be regulated. If these measures and a change of air and scene are insufficient, Weir Mitchell's

method should be tried, with its various developments of isolation, nursing by strangers, absolute rest in bed, overfeeding, massage, and electricity. The patient should get about 5 ounces of milk every three hours to begin with. The quantity is increased in a few days to 10 ounces. Then solids are gradually added, until, in the course of a fortnight, the patient is taking three substantial meals of mixed solids, in addition to the milk, and perhaps soup besides. This overfeeding is continued for six or eight weeks, after which the dietary is gradually reduced to the normal, the massage is withdrawn, and the patient is allowed to get up. The treatment in the nursing-home should be followed by a change to some bracing locality.

Among drugs that may be used for the general state, in addition to iron and other tonics, are valerian, valerianate of zinc, and asafœtida.

In cases of paralysis, faradism may be employed. A paraplegic patient should have regular drill. In aphonia a laryngoscopic examination may be enough to effect a cure, but if this fails, the faradic current may be applied outside the larynx, while the patient is instructed to utter a sound; or a blister may be applied round the throat. Contracture should be treated by rubbing, passive movement, faradism, blistering, or the actual cautery. For anæsthesia, the faradic brush should be employed.

For convulsions, cold water may be poured over the head, or into the nostrils; smelling salts may be applied to the nose; and the skin may be faradised. Other measures are the inhalation of amyl nitrite, closure of the mouth and nostrils by a towel, and firm and prolonged pressure upon a hysterogenic zone. For troublesome fits, and for paroxysmal laryngeal spasm, Gowers recommends the hypodermic injection of apomorphine ($\frac{1}{16}$ to $\frac{1}{12}$ grain).

Some hysterical symptoms, and especially pains, are best disregarded as much as possible. If headache is very intense, potassium bromide with arsenic may be given thrice daily. If anorexia and vomiting are troublesome, the nasal or stomach tube and the rectal syringe may be required for feeding purposes.

xx. Hypochondriasis.

This is a chronic nervous disorder characterised by mental depression which is due to needless anxiety about the health. It is really a slight form of insanity akin to melancholia. The patient either fancies he has disease when he has none, or attaches an exaggerated importance to trifling symptoms. The disease is much more common in males than in females, and occurs chiefly in adults. The patient is constantly thinking of his health, inspecting his tongue and evacuations, talking of his ailments, and consulting doctors. Some patients constantly refer their complaints to the same organs, such as the stomach, liver, sexual organs, or brain ; whilst the fears of others have reference to different parts at different times. A trifling uneasiness about the stomach will suggest cancer. Occasional palpitation will point to heart disease. Constipation suggests serious obstruction of the bowels. A few nocturnal emissions are interpreted as certain forerunners of impotence. With his thoughts constantly directed towards himself, and bent on studying, and discovering further symptoms of, his supposed organic ailment, the patient naturally magnifies any little abnormal sensation, and this grows in intensity with the attention bestowed on it. Under such circumstances, with internal phenomena occupying so large a share of consciousness, it is natural that the consciousness of external things should be diminished, and that the patient should imagine that his memory is being lost.

Careful and repeated examination by the physician may reveal no disease whatever, or at most some trifling change, which is quite insufficient to justify the patient's anxiety.

The disease is specially apt to occur in persons with a family tendency to insanity, and in many cases there is or has been some bodily ailment which may be regarded as its exciting cause. Disturbances of the digestive system are among the most common causes. Though the mental depression is great, there is no suicidal tendency.

Prognosis.—Hypochondriasis, especially if depending on a neurotic heredity, may persist throughout life ; but the

acquired form may undergo improvement or cure. Occasionally it gives place to melancholia.

Treatment.—This includes the correction of any bodily error that can be discovered, such as dyspepsia, constipation, bleeding piles, varicocele, etc. Alcoholic, sexual, and other excesses must be stopped. Tonics and a change of scene, especially in good company, may be helpful. The patient should be encouraged to educate himself to disregard the symptoms which he has been in the habit of attributing to serious disease.

xxi. Neurasthenia

(Nervous Exhaustion).

Etiology.—Neurasthenia, or exhaustion of the nervous system, may be due to a congenital lack of staying power on the part of that system, and is sometimes inherited. In such cases the symptoms of a breakdown may first appear in childhood, at puberty, or at a later period. Most cases, however, are seen in persons in the prime of life. In many instances a distinct immediate cause for the breakdown can be recognised, such as overwork (especially mental overwork), insufficiency of sleep, anxiety, worry, or emotional shock. Influenza and other acute febrile diseases are powerful causes ; and certain chronic abdominal disorders, such as movable kidney and enteroptosis, are apt to be associated with neurasthenic symptoms. The disease may also be associated with the abuse of alcohol, tobacco, morphine and cocaine, partly as cause and partly as effect. The shock which results from an accident is a common and important cause, but this is considered separately under Traumatic Neurasthenia. In patients who have suffered from neurasthenia, relapses and recurrences are induced by quite trifling causes.

Pathology.—No changes have been found in the nervous system to account for the symptoms. There is no doubt, however, that these depend upon a lowering of the functional capacity of some of the highest centres.

Symptoms.—These are mental and physical, general and local ; but all kinds are not necessarily present in any particular case.

The ability to do mental or physical work is reduced, so that an attempt to perform it causes speedy exhaustion. Those who for many years derived pleasure from their daily toil now find the slightest effort irksome. Yet the neurasthenic may appear strong if the efforts by which they are tested are very brief. The power of attention is much impaired, and as a necessary consequence the memory suffers. With the impairment of attention and object-consciousness, the patient becomes introspective and self-conscious, a condition which may be the cause of intense distress.

Headache is very common, especially pain or pressure in the vertex or occiput, but it is not usually severe. Pain is often complained of in the back and legs, and may be associated with numbness, tingling or coldness. Vertigo and insomnia are also troublesome. The patient is apt to become irritable in disposition, to become ' thin-skinned,' and to imagine that he is misunderstood by others. Mental depression is the rule, and in a good many cases there is a recognisable degree of mental alienation in the form of melancholia. Indeed, the patient may commit suicide. There may be insomnia by night, and great restlessness of mind or body by day.

Some patients are the victims of particular fears, such as the fear of being in an open space (*agoraphobia*), of being in a confined place (*claustrophobia*), or of society (*anthropophobia*).

In many cases the phenomena tend to localise themselves in certain organs or regions of the body. If the patient has ever suffered from disease, injury or pain in any part, however trifling such trouble may have been, the neurasthenic pain or paræsthesia is particularly apt to attack the part that was so involved.

In some cases neurasthenia is at once seen in the countenance of the patient. There may be an aspect of weariness in the eyes, attributable in part to slight ptosis and to

fatigue of the ciliary and other ocular muscles (*asthenopia*), aggravated, it may be, by refractive error; and in part to slight mental confusion caused by inability to fix the attention, and perhaps by the consciousness of a disagreeable tendency towards flushing of the face. In other cases, however, no such peculiarity of physiognomy is observed. A few minutes' reading, especially if the eyes move rapidly over the printed matter, and if the illumination is not perfectly suitable, is enough to induce pain in the eyes and head, and exhaust the brain for the time being. Conversation, even with the most well-meaning friends, causes brain-fag very quickly. There may be disturbances of hearing, including noises in the ears or head.

Palpitation of the heart or abdominal aorta, tachycardia, intermittence of the heart's action, flushing, blushing, exaggerated pulsation of the arteries, pulsation in the capillaries, and profuse perspiration may be present.

Digestive symptoms are also common, including flatulence and constipation, though in some cases diarrhœa is met with.

Sexual neurasthenia is also common, and is characterised by irritability and weakness. Nocturnal emissions, emissions under the influence of mental excitement, loss of sexual desire, impotence or the dread of impotence, imperfect erection or premature ejaculation, and pain in the testis, are among the symptoms in men; while females may suffer from ovarian tenderness and dysmenorrhœa.

There is no definite paralysis, but a general lowering of muscular power. Tremor is frequently present, for instance in the hands, and especially in connection with voluntary movements. The knee-jerks may be exaggerated on the one hand, or so sluggish on the other that reinforcement is needed to bring them out.

Diagnosis.—In many cases this must depend wholly upon the subjective phenomena. Except, possibly, loss of the plantar reflex, there may be nothing whatever of an objective description to indicate that the patient is not perfectly healthy, or that he is not shamming. He must be thoroughly examined with a view to excluding organic

disease, such as cerebral tumour, insular sclerosis, malignant or other disease of the viscera, etc. If all these can be pretty confidently excluded, the history of the illness, and the causation, together with the subjective symptoms complained of, will usually make the diagnosis clear. The presence of optic atrophy, reflex iridoplegia or Babinski's sign points to something more than neurasthenia.

Hysteria commonly shows itself earlier in life than neurasthenia; the symptoms may set in and disappear quite abruptly; and there are likely to be at one time or another some of the more distinctive phenomena of hysteria, such as convulsions, paralysis, anæsthesia, *globus hystericus*, etc.

Hypochondriasis is not specially characterised by exhaustion of mind and body. The patient's attention is concentrated on one part of his body, and he magnifies the significance of some trivial departure from the absolutely normal. His disease is really a mental 'twist' or perversion.

In *myasthenia gravis* the power of a muscle is lost as the result of exercise, in a period of seconds or minutes, so that a movement performed at the beginning of the observation becomes impossible in the short time mentioned. The myasthenic reaction may be obtainable, and there may be some permanent weakness of muscles supplied by cranial nerves.

Prognosis.—If the exciting cause can be removed, recovery may be hoped for under judicious treatment, even though the symptoms have been present for years. Some cases, however, are very obstinate; and even in those that do well, the progress towards recovery is not continuous, but by 'ups and downs,' a fact of which the patient should be warned at an early stage of the treatment. Even under favourable circumstances, months or perhaps years may be needed for complete recovery. Since the best method of treatment is expensive, the prognosis is influenced to some extent by the pecuniary position of the sufferer.

Treatment.—Any recognisable cause must be removed. Regularity must be observed in eating, drinking and sleeping, and all excesses must be avoided. A daily bath is sometimes of good service. A sea voyage or a residence in

a bracing country region may be recommended, and tonics (except strychnine) should be tried at first. On account of the enfeeblement of object-consciousness, and the abnormal activity of subject-consciousness or introspection, it is most desirable that the patient should have not only a regular occupation, but also a hobby which will absorb his thoughts when he is not on duty and entice them away from himself. Unfortunately it is not easy for such a patient to procure a hobby when told to do so, but friends may possibly be able to assist in this direction. Where simpler measures fail, a course of Weir Mitchell's treatment should be carried out in all its strictness, with complete rest, complete isolation from friends in a strange house, systematic massage to take the place of muscular exercise, and systematic overfeeding. This treatment should extend over six or eight weeks, and should be followed by a change to some bracing region in the country, or by a sea voyage.

Among the drugs that may be found useful in the treatment of special symptoms of the disease are iron, arsenic, sodium bromide, valerian, and nitroglycerin. If, again, the arterial tension is low, digitalis with a mineral acid should be tried.

xxii. Traumatic Neuroses.

To this group of affections belong *railway brain* and *railway spine*, *traumatic neurasthenia* and *traumatic hysteria*.

Etiology.—Any injury, but especially such injuries as result from railway accidents, together with the associated mental shock, may be followed by the symptoms of well-marked *neurasthenia*. The apparent bodily injury may be very slight, or there may be none, and indeed general neurasthenic symptoms are all the more likely to occur when there is no important local lesion. Thus the individual gets a violent and unexpected shock, but is able to go home and resume his usual duties. After some hours or longer he feels his back stiff, and still later, perhaps after days or weeks, the general symptoms set in, such as headache, noises in the head, constant weakness, nervousness, etc.

In some cases the symptoms are those of *hysteria* rather than of neurasthenia. Thus there may be hemianæsthesia with involvement of the special senses.

Spinal irritation may persist long after other symptoms have passed away. This is a neuralgia of the spine which may involve a considerable portion of its length, and is associated with tenderness.

Diagnosis.—This may be extremely difficult. The symptoms are in the main those of non-traumatic neurasthenia, but the fact of the injury necessitates particular caution lest structural disease should be overlooked. The presence of optic atrophy, disturbance of the sphincters, girdle sensation, localised weakness and wasting of muscles, and a well-marked ankle clonus strongly suggest organic disease when they are present, even singly. Their absence is of much less value in the opposite direction.

Prognosis.—No prognosis can safely be given at first. The symptoms of traumatic neurasthenia may pass away completely after a period of rest and change, but even in apparently slight cases it is well to avoid forecasting the future until the symptoms have at least ceased to progress. The sooner symptoms set in, the better is the ultimate outlook in cases which are not speedily fatal from organic disease. An extraordinary degree of recovery may take place even from symptoms pointing to structural change. In many cases, however, recovery is incomplete.

Treatment.—This must begin with rest. According to the aspect presented by the case, the measures recommended for structural disease of the cord, for neurasthenia, or for hysteria should be employed. It is obvious that the worry and suspense arising from any unsettled claim for compensation for injury must tell against a speedy recovery.

xxiii. Periodic Paralysis

(Family Periodic Paralysis. Intermittent Paralysis).

Etiology.—The disease generally attacks more than one member of a family, although isolated cases are met with. It may run through several generations. It is most common

in the first thirty years of life. Both sexes suffer. It has been supposed that malaria is a cause. The attacks may come on when the patient is at rest, or after muscular effort, or after the ingestion of particular articles of diet.

Morbid Anatomy.—Degeneration has been found in portions of muscle removed during life, but no morbid changes have yet been found sufficient to account for the symptoms.

Pathology.—It has been supposed that the symptoms are due to auto-intoxication. The known facts of the disease, including the electrical condition of the muscles, point to the muscle fibres and motor nerve endings being attacked by the poison. It is possible that these structures are abnormally susceptible to the action of the toxin in the individuals who suffer.

Symptoms.—The attack begins in an apparently healthy individual, without obvious cause, and often during sleep. The legs suffer first, and afterwards the arms and trunk; but the diaphragm and the muscles supplied by the cranial nerves commonly escape. The palsy progresses from the proximal to the distal portions of the limbs, and usually attains its worst within twenty-four hours. Sensation, the sphincters, and the mental faculties are unaffected.

The paralysis is flaccid, and when the attack is at its height, mechanical, galvanic and faradic irritability are alike lost. The deep reflexes, and sometimes the superficial reflexes, are diminished or lost. Dilatation of the heart has been noted in the course of an attack.

After some hours, the paralysis passes off, the legs being last in regaining power. The various symptoms mentioned disappear, and within a day or two the patient seems perfectly well. The interval between the attacks is often a week or two, but may be as long as a year; or the recurrences may take place daily.

Prognosis.—Death has sometimes taken place in an attack, but this is quite exceptional, and the disease commonly passes off after middle life.

Treatment.—Quinine and potassium citrate are reported to have been beneficial in a few cases.

xxiv. Astasia-Abasia.

In this disease, the patient is unable to stand (*astasia*) or walk (*abasia*), though when the legs are tested in the sitting or recumbent posture the strength of the muscles is found to be good. There is no spasm, inco-ordination, or sensory disturbance.

The pathology is uncertain, but the condition is apparently a pure neurosis. Recovery is the rule.

Treatment should be carried out on the same general plan as for hysteria.

VASOMOTOR AND TROPHIC DISEASES.

i. Raynaud's Disease.[1]

In a typical case of this disease, three symptoms can be recognised : local syncope, local asphyxia or local cyanosis, and symmetrical gangrene ; but in any given case, one or even two of these phenomena may be absent.

Local syncope is seen in the familiar ' dead fingers,' and is due to an exaggeration of the vascular constriction which normally occurs under the influence of exposure to cold. The arterioles are contracted, and probably in many cases the venules also. The affected part is white and cold ; it feels numb ; and tactile sensation is impaired. The venules appear to relax before the arterioles, and this leads to the stage of *local cyanosis* or *local asphyxia*. The part is blue and sometimes becomes black. If there is no stage of local syncope, the condition may be due to spasm of the venules. If local cyanosis persists in intense degree for many hours or for days, portions of tissue, chiefly skin, are liable to undergo *necrosis*. In these severe cases, there may be terrible pain in the affected parts. The gangrene, however, is sometimes much more extensive.

Raynaud's disease is markedly paroxysmal in character.

The parts affected by ' Raynaud's phenomena ' are those

[1] A detailed account of this affection, with a bibliography, is given in the author's monograph (Glasgow, 1899). Raynaud first described the disease in his inaugural thesis, published in 1862.

which are most liable to cool by radiation, viz., the extremities of the limbs, the tip of the nose, and the margins of the ears.

Etiology.—Females appear to be more liable than males to the most severe types of the disease. The affection occurs chiefly in the first half of adult life. A hereditary tendency is sometimes present. The most important cause is exposure to cold, and for this reason occupation and season are influential. A single exposure may not only cause contraction of the vessels for the time being, but may produce so profound an impression on the vasomotor system that it remains for a long time morbidly sensitive to the influence of cold. Severe emotion and malaria are also causes. Tuberculosis, disease of the heart and bloodvessels, paroxysmal hæmoglobinuria, sclerodermia, and various neuroses may be associated with Raynaud's disease.

Pathology.—The disease appears to be a neurosis characterised by abnormal excitability of the vasomotor centres in the cerebro-spinal axis. Its phenomena may, however, be sometimes induced by a poison in the blood acting temporarily on the vasomotor mechanism.

Prognosis.—The disease does not cause death, but in severe cases entails great suffering. After one or many paroxysms of local cyanosis, with or without gangrene, it may subside partially or completely. In some instances the tendency appears to be lifelong, but even after suffering severely during several winters, a patient may hope to escape serious trouble in the future.

Treatment.—This aims at removing the vicious habit acquired by the vasomotor mechanism. Everything should be done to postpone the occurrence of a fresh paroxysm. Attention must therefore be given to clothing, diet, the water used for washing, the avoidance of undue exposure, exercise, and if need be medicines. Quinine should be tried in periodic and malarial cases. Vasomotor relaxants, such as nitroglycerin, deserve a trial, though they are often unsuccessful. Opium should be freely administered in cases of gangrene.

Friction of the cyanosed part should be employed if the

patient can stand it. The faradic or galvanic bath should also be regularly used. The bath is particularly satisfactory because a large extent of affected skin can thus be treated at one time, and because it allows the current to be applied to parts which the patient could not allow to be touched by an ordinary electrode. The affected portion of limb is placed in a basin of hot salt water in which the negative pole of the galvanic circuit is also placed. The positive pole is applied to the upper part of the limb or to the neck. The current should be the strongest that the patient can comfortably bear, and should be made and broken or reversed frequently. The application should be made daily. Faradism may be employed in a similar manner. Another useful measure is to apply an elastic bandage to the limb so as to drive the blood in it towards the trunk. After one or several minutes the bandage is removed, whereupon the blood tends to rush into the part, and cause flushing. This method also should be employed daily.

Amputation of fingers, etc., should not be hastily resorted to, since the extent to which apparently dead parts will ultimately recover is sometimes remarkable.

ii. Erythromelalgia

(Red Neuralgia).

This is a rare disease characterised by intense burning pain, a livid red colour and local elevation of temperature in the peripheral part of one or more limbs. The skin becomes tense, hyperæsthetic, and slightly swollen. The veins are enlarged, and the arteries throb excessively. The condition is induced, or if persistent, is much aggravated by letting the part hang down, or by exposing it to heat. Elevation, on the other hand, and usually also a cool atmosphere, remove it for the time being, or at least relieve it.

Erythromelalgia may occur as an isolated condition, or in association with structural disease of the nerves or spinal cord. The most constant local change appears to be chronic endarteritis, but this may be a secondary lesion. Some cases recover, while others remain unchanged or become slowly worse. The pathology is obscure.

The treatment consists in prolonged rest with elevation of the part and the local application of cooling and anodyne agents.

iii. Angioneurotic Œdema

(Acute Circumscribed Œdema).

This condition is characterised by the sudden appearance from time to time of transient localised, œdematous swellings. These occur on the eyelids and other parts of the face, the hands, genitals, etc. By involving the larynx, they are occasionally a cause of death. The attacks may recur every few weeks, or much more frequently, and are commonly associated with gastro-intestinal disturbance. The disease may be inherited through several generations, and is perhaps akin to urticaria. No satisfactory treatment is known, though Osler speaks favourably of strychnine in large doses, and of nitroglycerin administered during a long period.

iv. Facial Hemiatrophy

(Facial Atrophy).

This rare disease may follow an injury to the face, or a specific fever ; or it may be inherited. As a rule, however, no cause can be recognised. It generally begins before puberty, and females are more liable than males. A gradual diminution in size of one side of the face may be the first symptom noticed ; or a patch of skin may become pale and depressed, and this condition may spread over the entire half of the face. The bones are diminished in size, and the wasting of the subcutaneous tissue allows the skin to become drawn in. The eyebrows are lost or become grey, and the teeth may become loose. Sometimes the tongue shares in the atrophy, through diminution of its interstitial tissue. The facial muscles do not degenerate, though they lose in bulk through loss of their interstitial tissue ; but the muscles supplied by the fifth nerve have been found atrophied. Sensation is not impaired, but there may be neuralgic pains. In rare instances, the disease becomes bilateral. The

pathology of the condition is obscure, but in more than one case an interstitial neuritis of the fifth nerve has been found.

Facial hemiatrophy is incurable, but it is not dangerous to life or health.

v. Acromegaly.[1]

Definition.—A chronic disease characterised by enlargement of the bones, especially of the hands, feet and face, and usually associated with changes in the pituitary body.

Etiology.—The disease is more common in females than in males, and generally commences in the first half of adult life.

Morbid Anatomy.—In almost every case, there are changes in the pituitary body, the lesion being very often a tumour. Thus in a case examined by myself, there was a round-celled sarcoma of the pituitary body, which had led to erosion of the neighbouring bone, deepening of the middle fossa of the base, and extensive destruction of the crura cerebri.[2] The thyroid gland may be enlarged or atrophied, and the thymus may be persistent or even enlarged.

Pathology.—It has been supposed that acromegaly is due to loss or perversion of the assumed internal secretion of the pituitary body.

Symptoms.—The bones of the hands and feet are much enlarged. The bones of the face also grow in size, and in particular the lower jaw, but the cranium is not much altered. The cartilages of the nose, eyelids, ears, and larynx may be thickened. The tongue is enlarged. Spinal curvature (kyphosis) develops. Amenorrhœa is an important early symptom. Temporal hemianopia and ultimately blindness may result from pressure on the optic chiasm. Similarly paralysis of the limbs may result from pressure on the crura cerebri. Optic atrophy is common. There may be intense headache, and, as in some other cases of intracranial tumour, this may be associated with the

[1] Described by Dr. Pierre Marie in 1885.

[2] Johnston and Monro, *Glasgow Medical Journal*, August, 1898, p. 112. It is to be noted that disease of the pituitary body does not necessarily give rise to the phenomena of acromegaly.

'hydrocephalic cry.' The disease commonly lasts for years. Myxœdema, exophthalmic goitre, and pancreatic glycosuria are occasional complications.

Treatment.—This must consist chiefly in good nursing when the patient becomes helpless, and in giving remedies for the relief of pain.

vi. Hypertrophic Pulmonary Osteo-arthropathy.[1]

This is a chronic condition characterised by enlargement of the hands, wrists, feet, and ankles, but without change in the head. The distal phalanges are bulbous, and the nails are curved. The disease is met with chiefly in the subjects of chronic lung disease, bronchitis, empyema, etc., and it may undergo considerable improvement if the primary condition is cured. It has been supposed that the tissue changes in the extremities are due to poisons absorbed from the seat of disease in the lungs, giving rise to a sluggish periostitis. Another theory is that they are tubercular.

vii. Osteitis Deformans.

In this rare disease there are changes in the long bones and in the cranium. The long bones become enlarged and softened, so that the legs become curved and the stature is shortened. The clavicles become prominent, a cervico-dorsal kyphosis develops, and the cranium undergoes enlargement, while the face is unchanged.

Diagnosis.—So far as the limbs are concerned, the long bones suffer most in *osteitis deformans*, the hands and feet in *acromegaly*, and the wrists and ankles with the hands and feet in *osteo-arthropathy*. The face suffers in *acromegaly*, while the cranium almost escapes. The cranium suffers in *osteitis deformans*, while the face escapes. In *osteo-arthropathy*, both face and cranium escape.

Osteitis deformans runs a chronic course. No satisfactory treatment is as yet available.

[1] Described by Dr. Pierre Marie in 1889.

SECTION IX

DISEASES OF THE MUSCLES

i. Thomsen's Disease[1]

(Myotonia. Myotonia Congenita. Transient or Family Myotonia).

Etiology.—This disease is often hereditary, and affects several members of a family. Thus Thomsen, after whom it is named, is himself a sufferer, and it has existed in his family for at least five generations. The affection is met with in both sexes.

Morbid Anatomy and Pathology.—In one case which was examined after death, no changes were found in the medulla, cord, or peripheral nerves. Hypertrophy of the muscle fibres and increase in the nuclei of the sarcolemma have been observed. It has been commonly held that the disease is a functional disorder of the muscular tissue; but Gowers has suggested that the morbid functional state may be shared by, or even primarily seated in, the motor cells of the cord or cerebral cortex. The disease is perhaps due to developmental defect, but the theory of auto-intoxication of the muscle tissue has been put forward.

Symptoms.—These are usually first noted in childhood, and are then due to a congenital tendency; but in a few cases the disease appears to be acquired at or after puberty. The characteristic feature of the affection is tonic spasm of the muscles, which occurs on first attempting to use them after

[1] Thomsen's account of the disease was published in 1876, and was the means of making it well known, but he was not the first to describe it.

a period of rest (' intention-spasm '). With repeated efforts, the rigidity at last passes off, and the voluntary use of the muscles can thereafter be easily kept up. The legs usually suffer most, but the muscles of the arms, and those of mastication, deglutition, and even micturition and defæcation, may be hampered by the same kind of spasm.

The affected muscles are often enlarged and are sometimes, but not always, stronger than is normal. They are very sensitive to mechanical irritation, so that a slight blow causes a localised swelling of the muscle. They also show the *myotonic reaction* :—faradism, and still more galvanism, cause a contraction which lasts abnormally long ; anodic contraction is often obtained as readily as cathodic ; and while the galvanic current is flowing, wave-like contractions may be seen to pass from the cathode to the anode.

Sensation and the superficial reflexes are normal. The deep reflexes are normal or increased.

Prognosis.—When the disease has fully developed, it generally persists throughout life. Some cases remain slight, even though they begin at an early age.

Treatment. — No cure for the disease is known, but Thomsen thinks that an active mode of life tends to lessen the annoyance which the condition entails.

ii. Myositis.

Polymyositis (*dermatomyositis*) is a rare disease characterised by acute interstitial inflammation of muscles, with erythema of the overlying skin and œdema of the subcutaneous tissue. The nerves may be slightly involved. The condition is symmetrical and probably due to a poison in the blood, as in the case of polyneuritis. The symptoms include pyrexia, weakness, and severe pain in the muscles. Death may result from involvement of the muscles of deglutition and respiration. The affected muscles show hyaline degeneration of their fibres, and round-celled infiltration of their interstitial tissue. Care should be taken to exclude trichinosis.

Myositis ossificans is a rare disease in which the muscles are the seat of a slow progressive fibrosis, and ultimately,

in many places, of ossification. The disease begins in the muscles of the back, and if, as occasionally happens, it spreads to the limbs, the whole body may, after a long time, become rigid like a statue. The face and diaphragm usually escape. The affection begins in childhood, and cannot be arrested by any known method of treatment.

Neuromyositis is an affection most often confined to one arm, and characterised by tenderness of the muscles, but still more by pain on contraction. It is most common in elderly people, but may occur in young subjects as a sequel of acute rheumatism.

iii. Muscular Dystrophy

(Progressive Muscular Dystrophy. Myopathy. Progressive Atrophic Myopathy. Primary Progressive Myopathy. Idiopathic Muscular Atrophy. Muscular Abiotrophy).

Pathology.—Muscular dystrophy is the collective name given to a disease or group of diseases which depend on a defective power of development in the tissue from which the voluntary muscles are derived. The developmental power is sufficient for the growth and activity of the muscles for some time after birth, and even, it may be, for many years ; but it fails at length, and the muscular fibres gradually succumb. Strictly speaking, therefore, the disease is of congenital origin, and this fact accounts for two features by which it is characterised ; namely, first, a tendency to affect several members of a family (though isolated cases are also met with) ; and secondly, a tendency to begin before maturity is reached.

While the nutrition of the muscle fibres is failing, the interstitial tissue may behave in various ways. In some cases it increases in amount to such an extent as to compensate for the loss of bulk of the muscular tissue ; in other cases, any increase it may undergo is insufficient for this purpose ; while in yet other cases, as Gowers puts it, the interstitial growth attains a special luxuriance. In the last variety, fat cells develop in the hypertrophied fibrous tissue,

and the muscle as a whole, in spite of the wasting of its contractile fibres, is increased in bulk. In every case, however, the primary change is degeneration and atrophy of the muscular fibres.[1] It is true that some of these fibres may at first appear to be abnormally large, but many are small, and in the long-run atrophy tends to overtake them all. The wasting is naturally accompanied by weakness, and according to the change which takes place in the interstitial tissue, the weak muscle is of normal, of diminished, or of increased size. Even, however, where the interstitial growth is luxuriant in the earlier stages of muscular atrophy, that luxuriance is not maintained; and when the muscle fibres have undergone much wasting, the fibrous tissue also fails, so that in advanced stages very little enlargement remains even in parts where at one time it was conspicuous.

Morbid Anatomy.—The spinal cord and the nerves have been repeatedly found to be normal, even in cases of long duration and severe character. Slight changes have been discovered in a few instances, but these are attributed by Gowers to the prolonged inaction of the cord and the spinal curvature.

Etiology.—The tendency to appear in several members of a family has been already alluded to. Moreover, the disease may run through several generations. In the case of the pseudo-hypertrophic variety, the usual, though not invariable arrangement is that the males suffer from the disease, while the females transmit it. In the other varieties, however, the sexes suffer almost equally. The age will be alluded to in connection with the different types of the disease.

Varieties.—Three principal types of muscular dystrophy require separate mention, but it must be borne in mind

[1] This teaching of Gowers seems the most satisfactory explanation of the facts It has been suggested, however, that the congenital defect causes the increase of the fat and fibrous tissue, and that these latter cause pressure-atrophy of the muscle fibres. According to another and less improbable theory, some unknown trophic disorder influences the muscular tissue and the interstitial tissue simultaneously.

that there is no definite line of demarcation between them.[1]

1. Pseudo-hypertrophic Muscular Paralysis.—According to Gowers, this disease is from four to seven times as common in males as in females, and when it occurs in females, it is slighter in degree and later in development than in males. In one third of the cases, the symptoms are first noticed when the child tries to walk, and in another third at the age of from four to six years.

The weakness or awkward use of the lower limbs first attracts attention. The child has a difficulty in going upstairs, falls readily, and has great trouble in rising again. Yet on examination, the weakened muscles appear to be well nourished and probably indeed abnormally large. Other muscles may be found to be wasted without having ever been enlarged, whilst at a later stage the muscles which were at first enlarged may become wasted.

The muscles which are most apt to be enlarged are those of the calves, and next to these the infraspinati. Other muscles which are often changed in this way are the extensors of the knee, in whole or in part, the glutei, the lumbar muscles, the supraspinatus, the deltoid, and to a less extent the triceps and biceps.

The tendency to atrophy is shown in a remarkable degree by the latissimus dorsi and the lower part of the pectoralis major. The serratus magnus, the biceps, the flexors of the hip and the flexors of the knee may also be wasted.

Other muscles escape, viz., the intrinsic muscles of the hand, and usually the trapezius and rhomboids, and the muscles of the face, neck, and forearm.

One of the most characteristic results of the weakness in the extensors of the knees and hips is the mode of rising from the floor to the erect posture. The patient gets on to his hands and knees, and then extends the latter so that he rests on his hands and toes. He next moves his hands

[1] Under the title of 'Distal Myopathy' Gowers has described a variety in which the distal, but not the proximal parts of the limbs are involved, the upper part of the face being also affected (*British Medical Journal*, July 12, 1902, pp. 90, 91).

gradually nearer to his toes, and then suddenly transfers first one hand and then the other from the ground to the thigh just above the knee. Thereafter he gradually moves each hand further and further up the corresponding thigh, till at last, by suddenly throwing his shoulders back, he jerks himself into the erect posture.

The weakness in the calf-muscles is seen in the inability to stand on tiptoe. The weakness of the spinal muscles is manifested in the development of spinal curvature. In the sitting posture, there may be kyphosis involving the whole length of the spine ; and in the erect posture, there is lordosis in the lumbar region with compensatory kyphosis higher up. The patient stands with his feet separated, and his shoulders thrown back. His gait is waddling.

The knee-jerks and cutaneous reflexes diminish and disappear just as the muscles on which they depend become feeble. Similarly the response of the muscles both to galvanism and to faradism is gradually lost as the muscular fibres disappear. Contraction of the fibrous tissue in the affected calf-muscles produces talipes equinus ; but at a late stage contracture may develop in relatively strong muscles owing to loss of opposition of the feeble muscles. Lateral curvature of the spine may appear as a later deformity in consequence of the weakness of the muscles of the back. Fibrillary tremor does not occur in the affected muscles. Sensation and the sphincters escape.

Diagnosis.—The occurrence of the disease in early life, and in several members of a family, is very suggestive of muscular dystrophy. As Gowers has pointed out, the combination of enlargement of the infraspinatus with wasting of the latissimus dorsi and the lower part of the pectoralis major points strongly to pseudo-hypertrophic paralysis. The enlargement of the calf-muscles, and the mode of rising from the floor, are also important. *Cerebral diplegia* is easily distinguished by the muscular spasm, the exaggerated knee-jerks, and the absence of wasting in the latissimus and pectoralis.

Prognosis.—The disease is a progressive one and gives rise to increasing disability. When the patient becomes unable

to use the muscles voluntarily, the weakness and wasting make rapid progress. The patient seldom attains the age of twenty, death being commonly due to bronchitis or pneumonia. In a few of the anomalous cases which begin late, the disease becomes stationary before reaching its last stage.

Treatment.—Drugs cannot arrest the disease, but it is justifiable to give drugs of the class of nervine tonics. It is doubtful if electricity and massage are by themselves of any great use ; but voluntary exercise of the muscles is of immense service in retarding the progress of the affection, and should be regularly carried out. Fatigue, however, must be avoided. Tenotomy should be carried out in every case where it will allow the continued use of muscles which otherwise would fall into disuse. In advanced stages, great care must be taken to avert pulmonary disease.

2. Juvenile Type of Idiopathic Muscular Atrophy (Erb).—This variety of muscular dystrophy generally shows itself about puberty, and usually affects individuals of both sexes in a family. The wasting begins in the muscles of the upper arm and shoulder girdle, and afterwards attacks those of the trunk, pelvic girdle and thigh. Among the muscles most liable to suffer are the biceps, triceps, supinator longus, pectoralis major, latissimus, serratus, trapezius, trunk muscles, glutei and quadriceps extensor. The intrinsic muscles of the hand, the muscles of the face, and, as a rule, the deltoid, supraspinatus and infraspinatus, escape. Spinal curvature is one of the varied results of this disease.

Diagnosis.—The commencement in early life and the occurrence in several members of a family make *spinal muscular atrophy* improbable. The escape of the intrinsic muscles of the hand and of the deltoid, and the absence of fibrillary tremor, suggest muscular dystrophy and not spinal muscular atrophy. When the trapezius is affected, as is frequently the case, it suffers in its whole extent ; whereas in progressive spinal muscular atrophy, its upper part escapes.

Prognosis.—The prognosis is not nearly so grave in this disease as in pseudo-hypertrophic paralysis, and the patient may survive for many years.

Treatment.—This is similar to that which was recommended for the pseudo-hypertrophic disease.

3. Facio-scapulo-humeral type of Idiopathic Muscular Atrophy (Landouzy and Déjérine) or Infantile Type (Duchenne).—This variety may commence in childhood or at puberty, and usually affects first the orbicularis palpebrarum and orbicularis oris. It may spread to almost all the muscles of the face, producing the 'myopathic facies,' but it spares the ocular muscles and the tongue. It also extends to the shoulder girdle and upper arm, and to the trunk and lower limbs.

Diagnosis.—What has been said with regard to the diagnosis of the other types of muscular dystrophy is to a great extent applicable here. When the orbicularis oris and orbicularis palpebrarum are affected, the absence of paralysis of the tongue and of the ocular muscles respectively points against disease of the twelfth and third nuclei.

Prognosis.—This is less serious than in pseudo-hypertrophic paralysis. Cases which begin in childhood are less favourable than those which begin after puberty.

Treatment.—This is the same as for the other varieties of muscular dystrophy.

iv. Peroneal Type of Muscular Atrophy

(Peroneal Type of Family Amyotrophy. Neuritic Muscular Atrophy. Progressive Neural Muscular Atrophy).

Etiology.—This disease, like muscular dystrophy, generally begins in early life, and may also run in families and be inherited. But it differs from muscular dystrophy in the muscles which it attacks, and in sometimes following measles and being associated with evidences of neuritis. It is more common in males than in females.

Symptoms.—The wasting is first noticed in the long extensors of the toes or in the peronei, though it is possible that it may have existed still earlier in the intrinsic muscles of the foot. The atrophy tends to spread to the anterior tibial muscles, and perhaps to the calf-muscles, so that the

leg is wasted below the knee. In the course of some years it invades the intrinsic muscles of the hand, and afterwards those of the forearm, so that the distal halves of the four limbs are now wasted. Later on the atrophy may even involve the proximal segments of the limbs and the limb-girdles. The face does not suffer. Double club-foot (*talipes varus* or *pes cavus*) is very apt to occur. The atrophy is often, but not invariably symmetrical. Fibrillary contractions and the reaction of degeneration are occasionally present. There may also be pain and impairment of sensation.

Pathology and Morbid Anatomy.—The pathology of this disease is obscure, but peripheral neuritis, and also sclerosis of the posterior columns, and to a less extent of the lateral columns of the spinal cord, have occasionally been observed.

Diagnosis.—The peroneal type of muscular atrophy is distinguished from *chronic spinal muscular atrophy* by the age of onset, the distribution of the wasting, and the club-feet. It is distinguished from *muscular dystrophy* by the distribution of the wasting, and the frequent presence of fibrillary twitching.

Prognosis.—Cure cannot be expected, but arrest of the disease may take place at any stage, either temporarily or permanently.

Treatment.—No cure is known, but strychnine should be given hypodermically, and massage and galvanism should also have a fair trial.

v. Arthritic Muscular Atrophy.

When a joint is the seat of either acute or chronic inflammation, the muscles which act upon it, and especially the extensors, undergo wasting. The mechanism appears to be a reflex one, the trophic cells in the cord which preside over the nutrition of the muscles being influenced by afferent impulses from the diseased joint.

The wasting may persist for a considerable time after the joint has recovered, and in that case, electricity and massage should be employed locally, while strychnine is to be administered internally.

SECTION X

DISEASES OF THE SKIN

ELEMENTARY LESIONS.

In cases of skin disease, the elementary lesions are those which, taken collectively, constitute the eruption. They are either primary or secondary. *Primary lesions* are the first signs of disease at the part where they are observed. *Secondary lesions* follow, and are secondary to other lesions. Certain lesions, such as hæmorrhages and macules, may be either primary or secondary.

Macules (*maculæ*) are spots or stains due to pigmentation, as in freckles or after hæmorrhage. They do not disappear when pressure is applied to them, and in this way redness due to staining may be distinguished from redness due to excessive fulness of the cutaneous vessels. Macules may be primary, as in freckles (lentigo); or secondary, as in the copper-coloured pigmentation often left by syphilitic lesions, and the staining which follows an ecchymosis.

Papules (*papulæ*) or pimples are little solid elevations of the size of a pin-head or rather larger.

Nodules (*nodulæ*), formerly called tubercles, are solid elevations of the skin of a larger size than papules.

Wheals (*pomphi*[1]) are rounded elevations due to acute inflammatory œdema, and often of very brief duration. In many cases the centre is pale owing to compression of the bloodvessels by the exudation, while the peripheral portion is red. This lesion is seen in nettlerash (urticaria), and after stinging by nettles.

[1] πομφός = blister.

Vesicles (*vesiculæ*) are very small blisters with clear liquid contents. The smaller sizes of vesicles such as are seen in sudamina are described as *miliary*. Larger ones, such as are seen in herpes zoster, are called *phlyctenular*.

Blebs (*bullæ*) or blisters are much larger than vesicles, but vary greatly in size. They may be tense or flaccid. Their contents may be serous, bloody, or purulent. Bullæ are specially characteristic of, though not confined to, pemphigus.

Pustules (*pustulæ*) are like vesicles, but have purulent contents.

Hæmorrhages vary greatly in size, and undergo changes in colour with the lapse of time. Small ones which resemble flea-bites are called *petechiæ*. Larger patches are called *ecchymoses*. Primary hæmorrhages are seen in purpura simplex. Secondary hæmorrhages occur in the spots of typhus fever, and often in the lesions of erythema multiforme.

Excoriations (*excoriationes*) or abrasions result from loss of a greater or less part of the thickness of the epidermis. The raw surface pours out a serous fluid which dries into crusts. No scar is left.

Ulcers (*ulcera*) or sores result from suppuration, or from necrosis of tissue. They involve the true skin, and sometimes the deeper tissues ; and when they heal, scars remain.

Scars (*cicatrices*) consist of fibrous tissue covered by epithelium. The true skin, hair follicles, and glands are absent. Scars are often red or livid when recent, but they ultimately become white.

Fissures (*rimæ*, *rhagades*), chaps or hacks, are apt to occur where the elasticity of the skin is lost, especially if the part is always moving, as in the case of the lips or the back of the hands in cold weather.

Scales (*squamæ*) consist mainly of masses of epithelial cells thrown off by an abnormal kind of desquamation. When the scales are very fine, as in tinea versicolor, the desquamation is described as *farinaceous ;* when they are branny, as in measles, it is *furfuraceous ;* when they are very large, as on the hands after scarlet fever, it is *membranaceous*.

Crusts (*crustæ*) or scabs consist of dried secretion, often mixed with epithelial cells and dust. The secretion may be serous, bloody, or purulent.

Hyperæmia or congestion may be active or passive. *Active* hyperæmia may be due to vasomotor relaxation, or it may be the early stage of inflammation. The skin is likely to be bright-red in colour. In *passive* hyperæmia the circulation is abnormally sluggish, and the skin is livid. In both forms pressure on the skin causes local pallor ; when the pressure is removed, the colour returns quickly in active hyperæmia, but only slowly in the passive variety.

CLASSIFICATION.

A convenient practical classification is that adopted by McCall Anderson in his 'Treatise on Diseases of the Skin'[1] ; but only a general outline of the arrangement need be given here :

A. Functional Affections.

I. Affections of the skin.
II. Affections of the hair.
III. Affections of the sebaceous glands.
IV. Affections of the sudoriparous glands.

B. Organic Affections.

I. Inflammations.
II. New formations and tumours.
III. Hæmorrhages.
IV. Diseases produced by uniform causes.
  (*a*) Parasitic affections.
    (1) Due to vegetable parasites.
    (2) Due to animal parasites.
  (*b*) Syphilitic affections.
  (*c*) Tubercular affections.
  (*d*) Eruptive fevers.

Of the diseases which are included in one or other of the groups above named, many have been already described.

[1] Second Edition, 1894.

Thus purpura, which belongs to the hæmorrhages, is dealt with in Section IV. (p. 358). Cutaneous affections due to animal parasites will be considered along with other affections of similar origin in Section XII. Syphilitic and tubercular affections, and the eruptive fevers, have been described in Section I.

A. FUNCTIONAL AFFECTIONS.

i. Affections of the Skin.

1. Pruritus.

Pruritus is a functional disorder of the skin characterised simply by itching, without any eruption unless that due to scratching. It may be localised or general.

The *localised* form includes *pruritus ani* and *pruritus genitalium*. Pruritus ani may be caused in children by intestinal worms. In elderly people it may be due to congestion of the rectum, but it seems more frequently to be a pure neurosis. Pruritus of the genitals is especially common in women, and may be a source of intense suffering. It may be connected with glycosuria, and whenever it is complained of, the urine ought to be examined for sugar. The itching may, however, be reflex, or due to eczema or some other cause.

Generalised pruritus is frequently a phenomenon of jaundice. It may also be due to cold weather (*pruritus hiemalis*), and it sometimes occurs in elderly people as a persistent affection without obvious cause (*pruritus senilis*). It is occasionally induced by the taking of drugs, such as opium.

Diagnosis and Treatment.—It is necessary first to exclude scabies, pediculosis, urticaria, and all other organic causes of itching. Glycosuria, if present, must have appropriate treatment. If none of these conditions is present, any local or reflex disorder which might cause pruritus must be removed, if possible. If the itching persists, nerve tonics should be tried, such as strychnine, arsenic, and phosphorus ; and nerve sedatives such as valerian, atropine and phena-

zone. Simple warm baths, bran baths, and alkaline baths should be used, and pilocarpine may occasionally be given hypodermically. The affected parts should be sponged with a lotion of carbolic acid (2 per cent.), or of liquor picis carbonis or liquor carbonis detergens (½-1 per cent.). In troublesome pruritus of the genitals a solution of silver nitrate (1-4 per cent.) may be applied.

2. Atrophy of the Skin (Atrophia Cutis).

General atrophy of the skin takes place in old people along with atrophy of other tissues (*senile atrophy*).

A common form of atrophy is that which occurs over the abdomen and breasts of pregnant women, and over the abdomen in ascites. Wavy, scar-like lines (*lineæ albicantes*) with a whitish, mother-of-pearl appearance are thus produced. Similar lines (*striæ atrophicæ*) may develop on the buttocks, thighs and shoulders in either sex. A rapid development of cutaneous atrophy is occasionally witnessed in connection with an attack of acute disease.[1] In rare cases atrophy of the skin appears in spots (*maculæ atrophicæ*) instead of streaks. Streaks of atrophy may be felt as furrows even more easily than they are seen.

Morbid Anatomy.—In atrophy of the skin the papillary layer is completely wasted, and the epidermic, vascular and fatty tissues are reduced in bulk.

3. Pigmentary Anomalies.

Albinism is congenital absence of pigment from the skin, hair and eyes. The hair, therefore, is white, and the eyes are pink. The condition may run in families. The individual suffers from intolerance of light, and nystagmus may be present. Albinism is common among the lower animals.

Lentigo (*ephelis, freckles*) occurs specially in individuals with fair or red hair. It is most marked on exposed parts, and is intensified by exposure to a bright sun. Freckles are rarely seen in the first five years of life, but I have counted a large number on the face of a fair-haired boy of less than a

[1] Monro, *Brit. Med. Jour.*, May 27, 1905, p. 1144 *et seq.*

year and a half. The yellow or yellowish-brown spots are familiar to all.

Treatment.—Freckles are by no means disfiguring, and are sometimes the very reverse ; but if local treatment is desired, those measures may be tried which are recommended for chloasma (see below).

Chloasma occurs in larger patches than lentigo. It is a yellow or brown discoloration which appears on the forehead or neck of women, especially in pregnancy, but also in connection with various disturbances of the sexual organs (*chloasma uterinum*). This pigmentary disorder is not observed before puberty, and does not persist after the climacteric.

Treatment.—It may be sufficient to rectify any uterine or other disorder ; but if local measures are found necessary, the part may be sponged twice daily with a lotion of corrosive sublimate (gr. ii. in ʒi.), or of sodium hyposulphite (gr. xv. to lx. in ʒi.).

Melasma Caloricum (*ephelis ab igne*) is the dark-brown marbling which is seen on the front of the legs, and sometimes on the back of the wrists, of invalids who are constantly sitting in front of the fire.

Leucoderma (*vitiligo*) is characterised by the development of well-defined, rounded, white areas which differ from the rest of the skin in nothing except colour. They are usually surrounded by abnormally dark skin which gradually merges into the neighbouring normal skin. The patches vary greatly in size. The individual has thus a piebald appearance. When hairy parts are involved, the hair growing from non-pigmented skin is itself non-pigmented. There is no constitutional disturbance. The disease is common in dark races, but slight forms are frequently met with in this country.

Treatment.—No cure is known. Nervine tonics may be given, and an attempt may be made to modify the pigmentation of the skin by the local application of a mercurial lotion as recommended for chloasma.

ii. Affections of the Hair.

1. Alopecia (Baldness).

Baldness is in rare cases congenital. It is a common senile change (*alopecia senilis*), and often develops prematurely in males, being no doubt promoted by the pressure of the hat. Thinning of the hair (*alopecia simplex*) is a common result of acute fevers, and especially of syphilis.

Treatment.—The treatment of any of these forms of premature baldness includes the internal administration of tonics such as arsenic, strychnine and iron ; and the application of some preparation of cantharides as a local stimulant (*e.g.*, aceti cantharidis ℥ss, ol. ricini ʒii., sp. rosmarini ad ℥vi.). Some benefit may be obtained from free sweating induced every other night by a hot bath, a hot drink, and a pill of pilocarpine. The head should be frequently washed, dried and then brushed till the scalp is reddened.

2. Alopecia Areata (Tinea Decalvans. Porrigo Decalvans. Area Celsi).

This affection is characterised by a rapid shedding of all the hairs from well-defined patches of skin. These are left perfectly bare, smooth and white. The disease affects chiefly the scalp, but may attack other hairy parts. It is often very limited, but occasionally it involves every hair on the body. It attacks individuals of either sex and at any age.

Pathology.—Different opinions are held on this question. Some writers maintain that the disease is parasitic, but the evidence seems clearly to show that in many cases it is a trophoneurosis.

Diagnosis.—*Ringworm* may be difficult to distinguish from alopecia areata if the hairs fall out rapidly, and without breaking off as they usually do. In such cases it may be possible to detect a few stumps containing the ringworm fungus, and the disease may also have attacked other children in the same school or household.

Prognosis.—Recovery is the rule, but it is apt to be slow, and the new hair may long remain white.

Treatment.—This is similar to that of premature baldness, but the lotion may be stronger; *e.g.*, acetum cantharidis diluted with one or two parts of spirit. Or liniment of ammonia may be thoroughly rubbed in. The constitutional state should receive attention.

3. Hirsuties (Hypertrichosis).

Hirsuties is chiefly of importance as occurring in the form of a rudimentary moustache or beard in women.

If **treatment** is desired, the best is destruction of the papillæ of the hairs by electrolysis. The needle is connected with the negative pole, and is passed down to the bottom of the follicle. A current of from 1 to 3 milliampères is then allowed to flow for about a quarter of a minute.

4. Fragilitas Crinium and Trichorexis Nodosa.

Fragilitas crinium or abnormal brittleness of the hairs, and trichorexis nodosa or a beaded condition of the hairs require only to be mentioned in this work.

5. Canities (Greyness of the Hair).

Greyness of the hair is due to lack of pigment in the hair. It may be congenital or acquired, local or general. Complete congenital canities is seen in albinism. Greyness is a normal senile change, but often occurs prematurely. In rare cases it develops in a single night under the influence of intense emotion, and localised greyness may appear in connection with chronic neuralgia.

iii. Affections of the Sebaceous Glands.

1. Comedo (Blackhead. Grub. Worm).

Comedo is a hair follicle distended with inspissated sebaceous matter, in the midst of which there are epithelial cells, fine hairs, and sometimes *Acarus* (*demodex* or *steatozoon*) *folliculorum* or the *pimple-mite*. The duct of a sebaceous gland usually opens into the constricted neck of a hair follicle, and obstruction may thus easily take place.

Comedo is most common at and after puberty, and is seen chiefly on the face and upper part of the trunk. The lesion appears as a black and often slightly elevated spot. The thick sebum which may be squeezed out is whitish except at the outer end where it was exposed to dust.

Treatment.—The comedones must be removed by the finger-nails, by a watch-key, or by a special scoop. It is sometimes desirable to use in addition stimulating local measures, such as are employed in the treatment of acne. The affected parts should be thoroughly scrubbed once a day with soft soap and hot water.

2. Milium (Grutum. Strophulus Albidus).

Milium is a very small, round, white tumour, situated immediately under the cuticle, and due to accumulation of sebaceous matter inside a sebaceous gland. (In comedo, the accumulation is in the hair follicle.) Milium is most common on the eyelids, penis and scrotum.

If **treatment** is demanded, the superficial wall of each milium should be punctured, and the contents should then be squeezed out.

3. Seborrhœa (Steatorrhœa. Stearrhœa. Acne Sebacea).

Seborrhœa is an excessive secretion of sebaceous matter. Two principal forms have been recognised, but the tendency now, especially among the followers of Unna, is to regard the dry variety as a form of eczema.

(1) *Seborrhœa oleosa* (*steatorrhœa oleosa*) is most often seen on the face, and especially the nose, which looks and feels oily. Dust readily adheres to the greasy surface. According to Sabouraud, this condition, as well as acne and alopecia, is due to infection of the sebaceous follicles by a minute bacillus.

(2) *Seborrhœa sicca* (*steatorrhœa sicca*) is chiefly seen on the scalp (*pityriasis capitis*) and eyebrows, and is characterised by the production of thin whitish scales, which, as they dry, fall off in the form of scurf or *dandruff*. The hair

often falls from the affected part in great quantity. See later (Seborrhœic Eczema).

Seborrhœa also attacks the face (*S. faciei*). It likewise occurs under the prepuce in the male and between the labia in the female (*S. genitalium*); and when cleanliness is neglected, the sebaceous matter is apt to decompose and give off an offensive odour, in addition to irritating the parts.

Diagnosis.—The skin under the scales is less red than in ordinary *eczema*. In eczema the eruption is seldom confined to the hairy scalp, itching is present, and there is often a history of leeting.

In *lupus erythematosus*, the crusts are more adherent, there is more pain and irritation, and cicatricial changes are produced.

Treatment.—Appropriate measures must be used to remove anæmia, uterine disease, or any other general or organic disorder which may be present. In dry seborrhœa the crusts must be first removed by soaking with olive oil, and covering with flannel and gutta-percha tissue; the oil and softened crusts are washed off on the following day with soap and water. In both dry and moist forms, after the skin has been thoroughly cleansed, some greasy preparation such as zinc ointment is to be applied. The cleansing and anointing may be repeated daily.

iv. Affections of the Sweat Glands.

1. Anidrosis or absence of sudoriparous secretion, is observed in connection with various diseases such as diabetes, wasting conditions, and ichthyosis. In some individuals it exists as a natural peculiarity.

The **treatment** is generally that of the cause.

2. Hyperidrosis (*ephidrosis*) is a symptom of various diseases such as rheumatic fever and phthisis. It may accompany the critical fall of temperature in pneumonia, etc. In some individuals perspiration occurs chiefly or solely on one side of the body. Profuse perspiration frequently gives rise to cutaneous eruptions. Thus the perspiration accumulating between the layers of the epidermis gives rise to minute vesicles resembling droplets of water.

These are *sudamina*. If their contents become milky, the eruption is called *miliaria alba*. If the vesicles each become surrounded by a red inflammatory zone, the condition is *miliaria rubra*.

Perspiration may be localised either to one half of the body, or to symmetrical areas on the two sides; *e.g.*, the palms, soles, axillæ and genitals. At the flexures, excessive sweating may lead to inflammation of the skin.

Treatment.—In hyperidrosis, the general health must be attended to. Persons sweat more easily when they are run down than when they are in robust health. Atropine sulphate may be given each night (one or two pills, each containing $\frac{1}{80}$ grain) to check general sweating, and the skin should be sponged with tepid vinegar and water. Crocker recommends a level teaspoonful of precipitated sulphur in milk twice daily, combined, if need be, with astringents to prevent excessive purgation. Local sweating may be treated by the measures recommended for bromidrosis.

3. Bromidrosis (*osmidrosis*) is the secretion of sweat which, after secretion, acquires an offensive odour. The feet, and sometimes the axillæ, groins and perineum, are affected. The odour is due to the activity of micro-organisms.

Treatment.—Among the remedies which may be employed is a lotion containing 10 grains of tannic acid in 1 ounce of spirit. This is applied frequently and allowed to dry. In the case of the feet, frequent and thorough cleansing is important. The socks should be changed every day, and disinfected by boiling. The feet and interior of the socks should be thoroughly dusted with a powder of boracic and salicylic acids (ac. salicyl. gr. x., ac. boric ʒi.). The internal use of sulphur, as described under Hyperidrosis, may prove sufficient treatment of itself. Formalin solution (2 or 3 per cent.) is another remedy.

4. Chromidrosis or coloured perspiration and Hæmatidrosis[1] or bloody sweat are very rare.

[1] The Duke of Anjou died (June, 1584), 'in great torture, sweating blood from every pore.' Poison was suspected (Motley, 'Rise of the Dutch Republic,' part vi., chap. vi.).

B. ORGANIC AFFECTIONS.

i. Inflammations.

1. Erythema.

Erythema Simplex is characterised by redness, heat, and perhaps slight swelling. The redness disappears on pressure, and there is no moisture or definite infiltration. There is little or no constitutional disturbance. As the inflammation subsides, the heat and redness are replaced by itching and branny desquamation.

Treatment.—A dusting powder of zinc oxide or starch is sufficient local treatment.

Erythema Intertrigo is a form of erythema which develops where two surfaces are in contact, as in the axillæ, below pendulous mammæ, between the thighs, etc. Redness and itching are the principal features. The disease often passes on to eczema.

Treatment.—Cleanliness is important, and the parts should be dusted with powdered starch or zinc. If the skin is constantly being wetted, as by urine, it should be cleansed, dried and then anointed with vaselin.

Erythema Laeve is that variety of erythema which frequently develops in dropsical legs. It may pass on to extensive sloughing.

Erythema Paratrimma is the discoloured condition of skin which precedes the development of a bedsore.

Erythema Pernio (*chilblain*) is most common in children, and in adults with a sluggish circulation. It occurs chiefly in cold weather and affects the fingers, toes, and parts such as the ears, nose and cheeks which are readily cooled. The part is dusky-red, swollen, itchy, and somewhat painful. If neglected the chilblain may undergo ulceration.

Treatment.—Warm clothing, physical exercise, and in some cases cod-liver oil and tonics are indicated. The local applications to unbroken chilblains should be stimulating to the circulation, and may at the same time contain an anodyne. Liniment of camphor, of ammonia, or of mustard, or tincture of iodine, may be applied, with or without

liniment of belladonna. Ulcers should be treated on the usual surgical principles.

Erythema Multiforme (*Erythema exudativum multiforme, polymorphic erythema*) is a disease to which, in its very varied aspects, numerous designations have been applied. It is symmetrical, and affects the backs of the hands and forearms, and sometimes the dorsa of the feet and front of the legs, and even other parts in addition.

The eruption consists at first of red patches or papules (*Erythema papulatum*), or nodules (*E. tuberculatum*). The red discs tend to spread at their circumference and heal in the centre, so that rings are produced (*E. annulare*, *E. circinatum*, *E. iris*). A vesicle sometimes appears in the centre of each ring (*Herpes iris*), and occasionally bullæ develop (*E. bullosum*). If the rings grow till they meet and interrupt one another, wavy lines are the result (*E. gyratum*, *E. marginatum*). A single lesion may last for only a few days, but successive crops may prolong the attack for weeks or even months.

Subjective symptoms are seldom important ; there may be tenderness or itching, rheumatic-like pains, and slight pyrexia. The disease is most common in young adults, and is apt to recur in spring and autumn.

Pathology.—This form of cutaneous inflammation is possibly due to some toxin. There is hyperæmia, with exudation into the skin of serum and frequently of blood.

Treatment.—Scarcely any treatment is required, but in severe cases the patient should rest. Antirheumatic remedies, such as sodium salicylate, should be employed.

Erythema Nodosum (*dermatitis contusiformis*) is closely allied to, if not actually a variety of, erythema multiforme. The disease is usually ushered in by some constitutional disturbance, including pyrexia and rheumatic-like pains. The most common seat of eruption is the front of the legs, and the lesions consist of purplish-red, tender swellings, often of the size and form of a split almond, with their long axes corresponding to that of the limb. They go through the same changes of colour as an ordinary contusion, and for the same reason, namely, the occurrence of hæmorrhage.

Each node lasts for a few weeks, but there may be two or more crops. The disease is most common in young females, and tends to recur.

Treatment.—Unless the attack is severe, the patient need not keep to bed. If pyrexia and pain are considerable, salicylate of sodium should be given. Otherwise tonics such as quinine are indicated.

Purpura Rheumatica (*peliosis rheumatica, Schönlein's disease*), which has been alluded to in connection with purpura (p. 359), is to be regarded as a variety of erythema multiforme (*purpuric erythema*). Cutaneous hæmorrhages and articular symptoms are prominent features.

The diseases of the exudative erythema group, including purpura rheumatica, appear to be of infectious origin, although, like endocarditis and meningitis, they are not always due to the same infective agent. Thus we may find erythema, purpura, and arthritis associated with fatal valvular disease and angina pectoris in an adolescent ; or urticaria and purpura associated with aortic valve disease ; or erythema multiforme associated with pneumonia, or with the symptoms of polioencephalitis.[1]

Pityriasis Rosea (*P. maculata et circinata*) usually appears first on the front of the chest, and spreads thence to the rest of the trunk, the neck, and the proximal parts of the limbs. Rose-coloured spots appear which, as they grow at their circumference, fade at the centre, so that rings are produced. The rings may meet and give rise to gyrate figures. Slight pigmentation and fine desquamation may be observed. There may be slight itching when the patient is heated.

Diagnosis.—*Tinea circinata* is seldom so widely spread, and may, moreover, be distinguished by the presence of the fungus.

Psoriasis attacks by preference the extensor surfaces of the elbows and knees, and is characterised by a much coarser type of desquamation than is seen in pityriasis rosea.

In *syphilis*, the secondary eruption is polymorphous, and other evidences of the disease are likely to be present.

[1] *Brit. Med. Jour.*, May 27, 1905, pp. 1144-1146.

Prognosis.—Spontaneous recovery takes place in the course of a few weeks or months.

Treatment.—If the spots are few in number, they should be painted with tincture of iodine. If the eruption is more extensive, it should be treated locally with liquor picis carbonis in water (5 to 15 minims in 1 ounce).

Erythema Induratum (*Bazin's disease*) is characterised by the appearance of a series of nodules or indurated patches which run a slow course and are painless. The nodules are of the size of a pea or larger, and occur chiefly on the legs, especially below the fleshy mass of the calf-muscles. The nodules sometimes undergo slow absorption, but they frequently soften and give rise to ulceration. As they approach the surface, the overlying skin becomes livid in colour.

The eruption is generally present in both legs, and is most common in girls and young women.

Pathology.—The microscopic structure of the lesions, the results of inoculation-experiments, and the fact that the disease is commonly seen in scrofulous subjects, suggest that the condition is really tubercular.

Diagnosis.—In *erythema nodosum* the nodules are tender, the course is more definite and less protracted, and there is frequently some constitutional disturbance.

Syphilitic gummata may give rise to difficulty in diagnosis, but these are usually unilateral, and other evidences of syphilis may be present.

Prognosis.—The general health does not suffer, but the local affection may be very prolonged and obstinate.

Treatment.—Rest, elevation, and bandaging of the limb are indicated. In troublesome cases, scraping with a sharp spoon may be advisable. Tonics and other antitubercular remedies should be employed.

2. Eczema.

Eczema is a chronic or acute, non-contagious inflammation of the skin, accompanied by itching, and manifested by various lesions such as papules, vesicles and pustules, and often by serous exudation in the earlier stages, whilst in the later stages there is redness with scaliness.

Etiology.—Eczema occurs at all ages, but is specially common in infants and elderly people. Males are rather more liable than females. In many cases no distinct cause can be recognised, but there is often a constitutional tendency or some local irritation ; or both factors may be present.

Among the internal or constitutional causes may be mentioned hereditary tendency ; debility from anæmia, insufficient or improper food, etc. ; dyspepsia (as in the *lichen simplex*, *erythema strophulus*, or ' stomach rash ' of infants) ; imperfect excretion, either intestinal or urinary ; menstrual disorders, pregnancy, and prolonged lactation ; gout ; anxiety ; and shock, as from an accidental injury.

The local causes are numerous, and include irritation in connection with occupation (as in ' baker's itch,' ' bricklayer's itch,' and ' washerwoman's itch ') ; passive hyperæmia associated with varicose veins in the legs (' varicose eczema ') ; ointments, lotions, etc., applied for therapeutic purposes (as in unduly prolonged treatment of scabies by sulphur ointment) ; exposure to the hot sun and sea-breeze ; profuse sweating (as in the *lichen tropicus* or ' prickly heat ' of hot countries) ; the moisture and friction of cutaneous surfaces in contact with one another (*eczema intertrigo*) ; and parasites such as acari and pediculi.

Bacteriology.—There is good reason to believe that microbes are not necessary for the production of eczema ; but staphylococci and probably other pathogenic organisms readily settle in the diseased skin. To them we may attribute the obstinate character of some cases, the locally infective nature of the disease, and the production of its suppurative varieties.

Pathology.—In an acute case, there is dilatation of vessels with serous exudation in the papillary layer of the corium. The increasing infiltration of the corium and thickening of the deeper portions of the epidermis lead to the development of elevations which look like solid papules, though actually containing fluid. When the infiltration extends into the Malpighian layer of the epidermis, the upper layers of the cuticle are raised so as to form vesicles. The liquid contents

of these minute blisters are at first clear, but tend to become turbid owing to an accumulation of leucocytes within them. As a rule, however, this accumulation does not become so dense as to constitute pus. The vesicles readily rupture, either spontaneously or as a consequence of scratching, and the Malpighian layer is then exposed as a weeping surface. The vascular tufts become visible as small red points, from which exude droplets of a highly albuminous fluid.

Symptoms.—The eruption is polymorphous, a variety of lesions being often seen in one patient, and even, it may be, in one patch of eruption. Thus there may be erythema, papules, vesicles, pustules, excoriations, crusts and scales; but only one or two of these may be present, and the disease may be mistaken for a different affection. Special names have been employed to indicate the predominant feature of the disease in a given case, as *Eczema erythematosum* (erythematous), *E. papulosum* (with small red papules), *E. vesiculosum* (vesicular), *E. rubrum vel madidans* (weeping or leeting), *E. pustulosum vel impetiginosum* (pustular), *E. squamosum* (scaly), and *E. rimosum* (with fissures).

Eczema is generally symmetrical, unless due to a local cause. Whilst it may appear anywhere or everywhere, it shows a preference for flexor surfaces, and for the junctions of skin with mucous membrane. It is particularly common about the head in young children; while the legs are a common seat of the disease in elderly people, owing to varicose veins, etc.

Eczema is almost invariably associated with itching. The only exception to this rule is when the itching is masked by burning or actual pain, a condition which may be due to great acuteness of the inflammation or to the coexistence of fissures.

The inflamed skin is thicker and less elastic than normally, owing to infiltration with cells and serous exudation. The redness of the inflamed skin fades gradually into the colour of the neighbouring healthy skin.

In many acute cases, the skin pours out an exudation (*E. madidans*). This is usually serous, and has the property

of staining and stiffening linen, but is sometimes purulent owing to the development and rupture of pustules. Occasionally there is no appreciable discharge at any stage (*E. siccum*). The exudation, as it dries, forms crusts with the desquamated epithelium. The colour of the crusts varies according as the exudation is serous or purulent, and also depends on the admixture of blood, dust, or other substances.

Leeting or weeping is specially associated with acute eczema ; whereas scaliness, as distinguished from crust-formation, is more closely connected with the chronic disease.

Diagnosis.—An early stage of eczema might be mistaken for *erythema multiforme*, but the latter attacks the backs of the hands, whereas eczema is more likely to be on the flexor surface. In *erythema papulatum* and *erythema tuberculatum*, the spots are isolated, whereas in eczema the spots and redness are continuous and form considerable patches. Itching is much less marked in erythema than in eczema.

Erysipelas spreads over one limited area of skin, and the affected portion has a well-defined margin. The local pain and tenderness are considerable. Bullæ may be present, but there is no leeting or vesication. Severe constitutional symptoms accompany the local inflammation, and the disease ends spontaneously in less than two weeks.

Dry scaly eczema might be mistaken for *psoriasis*. The latter is especially apt to appear on the tips of the elbows and below the patellæ ; the scales are silvery or may be rendered so by scratching ; and itching may be absent. In eczema, the edges of the patches are less abrupt ; the scales are less firmly attached, and are seldom silvery ; the itching is greater ; the flexor surfaces are specially liable to suffer ; and there may be a history of leeting at an earlier period.

Scabies is really eczema produced by *Acarus scabiei*, and it is essential to make a correct diagnosis. The preference of the eruption for the lower part of the trunk, the thighs, the forearms, and the skin between the fingers is very suggestive ; and a history of contagion, and the discovery of canals made in the skin by the female parasite, are conclusive.

Sycosis, or *folliculitis barbæ*, may be difficult to distinguish from eczema of the face. In sycosis, the pustules at the hair-follicles are the essential feature; whereas in eczema the intervening skin is inflamed, and pustules, if present, are likely to be less numerous than in sycosis.

Prognosis.—Eczema rarely causes death, but is a source of much annoyance through the itching and disfigurement. If it is a result of the occupation by which the patient makes his living, the outlook is of course very serious. The acute and subacute forms can almost always be cured, but the disease is liable to recur.

Treatment.—In *acute* cases, rest in bed is important. At the outset a mercurial laxative should be given to clear out the digestive tract. The diet should be light, and tea, coffee, alcohol, oatmeal, green vegetables, and sweet and acid fruits should be avoided. Anæmia, struma, gout, kidney derangements, and other general and visceral diseases must be treated by appropriate remedies.

In *chronic* cases, the patient may benefit by moving about, by a change of air and scene, and by resorting to certain watering-places. In chronic, scaly eczema, and in pustular eczema, iron and arsenic may be beneficial.

In all kinds of eczema, it is important to avoid too frequent washing with water. Ordinary soap is, as a rule, hurtful.

The local treatment of *acute* eczema must be soothing. The inflamed part should be kept constantly wet with dressings of distilled water, lead lotion, or calamine and oxide of zinc lotion (℞ Calaminæ et zinci oxidi, āā ʒii.; glycerini. ʒss.; liq. calcis, ʒii.; aq. rosæ, ad ʒviii.—M.); or it may be dusted with oxide of zinc and covered with a starch poultice.

In *subacute* and *chronic* cases, it is often necessary to remove crusts before a remedy can come in contact with the skin. This is easily done by applying olive oil and then covering the crusts for some time with a poultice. The warmth and moisture soften the scabs and allow them to be lifted off without pain or bleeding. For cleansing purposes, soap must not be used, and even plain water is not so good as ordinary oatmeal gruel.

In *subacute* eczema, the therapeutic applications are inter-

mediate in character between the sedatives used for the acute disease and the stimulating measures suited for the chronic ailment. Thus in subacute cases, zinc ointment, or a lotion containing zinc oxide or calamine, or an ointment of the oleate of lead or bismuth, may be applied on strips of lint.

In very *chronic* cases, tarry preparations may be employed, but great caution is needed at the outset, in case the tar should prove too irritating. Another remedy is soft soap rubbed firmly into the skin night and morning, either undiluted or dissolved in one or two parts of water. If the infiltration is considerable, a stronger preparation of potash is available in the form of liquor potassæ, which may be painted over the affected patch once a day. If severe smarting is induced, warm water should be applied. When leeting eczema proves very obstinate, it is well to apply a parasiticide, such as white precipitate ointment, or solution of nitrate of silver (gr. xv. in ʒi.).

In eczema of the *head*, which is usually pustular, the hair should be cut short in children, and in adults also, if the condition is severe or chronic. The surface must be thoroughly cleansed by removal of crusts and washing with gruel, after which white precipitate ointment is to be applied. Benefit is often obtained by the constant wearing of a vulcanised rubber cap.

Eczema of the *margins of the eyelids* (*E. tarsi, tinea vel ophthalmia tarsi, marginal blepharitis*) may be treated by the local inunction of yellow oxide of mercury ointment (1 part in 1 or 2 parts of vaselin), or of white precipitate ointment (1 to 2 per cent.), or of an ointment containing 2 or 3 minims of creolin in 1 ounce of lanolin. All crusts must be carefully removed before the ointment is applied, and in troublesome cases the lashes should be epilated.

In eczema of the *hairy part of the face* (*E. pilare faciei*), the inflammation tends to involve the follicles and to give rise to pustules. The beard must be shaved off, and hairs which are surrounded by pustules should be epilated. After the acute stage is passed, all the hairs should be removed by epilation. Yellow oxide of mercury ointment should be well rubbed in.

In eczema of the *external auditory meatus*, the crusts and accumulated discharge must be got rid of by syringing, after they have been softened by the introduction of olive oil. The ear is then to be dried, and oxide of mercury ointment or, in persistent cases, nitrate of silver solution may be applied.

In eczema of the *hands*, a good result is sometimes obtained from the wearing of vulcanised indiarubber gloves. In troublesome cases of dry eczema of the palms, the scales should be removed by salicylic plaster, after which yellow oxide of mercury ointment may be applied. Arsenic or tar may be given internally.

In eczema of the *legs*, it is important to improve the local circulation, whether by elevation of the limbs, the wearing of elastic stockings, the disuse of garters, or otherwise.

3. Seborrhœic Eczema (Seborrhœic Dermatitis).

In ordinary dry seborrhœa of the scalp or common dandruff (p. 889), there is no redness or other obvious evidence of inflammation. Occasionally, however, on the scalp, and constantly when the disease spreads to other parts, inflammation accompanies the disordered sebaceous secretion. The eruption is at first follicular, but spreads in a serpiginous or creeping fashion. The skin is red and may be swollen and moist, or may be covered with greasy yellow scales or crusts. In children especially, the inflammation tends to spread from the scalp to the forehead, temples and ears; and similar changes may be found even on the trunk in adults.

The disease is probably parasitic, and due to a staphylococcus attacking the skin. Unna holds that seborrhœa sicca is simply a mild form, or the first stage, of seborrhœic eczema, and is accordingly inflammatory; and this view is pretty generally accepted, even though the clinical signs of inflammation may be absent.

Treatment.—If the eruption is very greasy, a lotion of liquor picis carbonis (1 to 2 per cent.) should be applied; but if it closely resembles eczema, it should be treated as if it were eczematous.

4. Impetigo Contagiosa.

This disease is chiefly seen among young children of the poorer classes. The predisposing cause is defective hygiene, and the exciting cause appears to be the inoculation of the *Streptococcus pyogenes* under the horny layer of the epidermis. Inoculation is effected by scratching, as in children with pediculosis capitis, who inoculate not only other parts of the head, but the margins of the mouth, nose, etc.; or by a fall or some other agency which produces trauma. The disease spreads rapidly among those children who are predisposed by their conditions of life.

Symptoms.—The disease is characterised by the development of flaccid vesicles or small bullæ whose contents soon become purulent. The vesicles speedily rupture, and their collapsed walls and their contents, together with desquamated epithelium, give rise to irregular greenish-yellow crusts, which look as if they were 'stuck on.' The related lymph glands undergo enlargement. In the case of the scalp, the hair may be much matted by the discharge. The constitutional disturbance is very slight. The crusts fall off after a few days, without leaving scars, though for a time the surface which is thus laid bare remains red and shiny. As the disease is auto-inoculable, numerous crops of lesions may develop.

Diagnosis.—The diagnosis has to be made from *eczema*. In impetigo, the individual vesicles or pustules are isolated and not surrounded by inflamed skin; the disease is usually non-symmetrical; there is little or no itching, unless eczema or pediculosis is also present; the affection is almost confined to poor children; the occiput is the most common primary seat; and the disease is inoculable.

Treatment.—This includes attention to the hygienic conditions, suitable food and tonics. Crusts must be removed, and pediculi must be destroyed. White precipitate ointment should then be applied. Scratching must be prevented.

Bockhart's Impetigo is a variety due to inoculation of the *Staphylococcus aureus* under the horny layer of the epidermis, usually at the mouth of a hair-follicle. It may

be inoculated in a primary way by friction, or may be a complication of impetigo contagiosa, or of the eruption caused by such irritants as turpentine. The lesions are situated chiefly round the hair-follicles, and are tensely filled with pus.

5. Urticaria (Nettlerash).

This disease is characterised by the development of wheals or pomphi which are accompanied by subjective sensations like those produced by the sting of a nettle. Contact with certain kinds of jelly-fish and of hairy caterpillars is also a local cause. In those individuals who are susceptible, an attack may be induced by the internal use of certain articles of food, such as shell-fish, strawberries, and mushrooms; or of certain drugs, such as copaiba and quinine. Digestive and genito-urinary disturbances may also be causes. Urticaria is apt to complicate other diseases of the skin which cause itching and lead to scratching. Much depends on individual peculiarity. In some people, stroking the skin with the finger-nail leads to urticarious swelling, so that letters can be traced upon the body (*factitious urticaria, dermography*).

The size and form of the wheals vary much, but a typical one is circular, elevated (from congestion and serous exudation), pale in the centre (from contraction of vessels), and red at the periphery (from dilatation of vessels). The wheals may develop suddenly all over the body (*acute urticaria*), or may appear in different parts in succession. The patient scratches and rubs his skin, and thereby aggravates the condition. An attack may last for hours or occasionally for a few days.

Varieties.—In some instances the attack is accompanied by fever (*U. febrilis*). In some cases attacks recur at short intervals for a long time (*U. perstans, chronic urticaria*). In urticaria which involves the face, there may be considerable œdema of the eyelids. *Urticaria papulosa* (*lichen urticatus*) is principally seen on the limbs of children, and is characterised by the development of papules as well as wheals. The former are very itchy and persist longer than

the wheals. The wheals are sometimes very large—for instance, as large as a hen's egg (*U. gigans, giant urticaria*). In *Quincke's disease*, or *acute circumscribed* or *wandering œdema*, the tissue of the orbit or of some other part of the face may swell so much as to constitute a large tumour; or there may be an extensive ill-defined area of superficial œdema in a limb. In these giant and diffuse varieties there is seldom itching, though burning may be present.

Urticaria pigmentosa is regarded by some as a distinct disease, though factitious urticaria is usually associated with it. It is a rare affection, which is characterised by persistent nodules with yellowish pigmentation of the skin. It first appears in early infancy, and tends to pass away spontaneously as puberty approaches. Itching may be severe, and may give rise to much scratching and secondary urticaria.

Treatment.—In the treatment of urticaria it is important to discover and remove any possible cause, whether this acts directly upon the outer surface of the skin, or acts reflexly, or acts on the skin after absorption from the alimentary canal. This may necessitate the adoption of a different kind of underclothing, the removal of round or thread worms from the intestine, the avoidance of certain articles of diet, etc.

After an attack has set in, if any unsuitable article of food is known to have been taken, an emetic and a purge should be given at the outset. In any case, for a time at least, the diet should be light, and a mixture may be ordered containing bismuth, soda and nux vomica. Quinine, sodium salicylate, salol, and antipyrin are drugs which may be found serviceable in some instances. In chronic cases, arsenic and alkalies may be given. To relieve the itching, warm alkaline baths may be employed, or the surface may be gently sponged with a 2 per cent. carbolic lotion, or with a drachm of liquor picis carbonis in a pint of tepid water. Scratching and rubbing must be avoided. Wright found that in the subjects of urticaria there is deficiency in the coagulability of the blood, and he gave calcium chloride (20 grains thrice daily) with some success.

6. Prurigo (Hebra's Prurigo).

This disease usually commences in infancy, and is characterised by the development of pale, subcuticular papules which at first can be better felt than seen. They are, however, intensely itchy, so that their tops are scratched off, and the drop of blood which exudes dries into a black crust. The lesions are mostly found on the trunk, buttocks, anterior aspect of the legs, and extensor aspect of the forearms. In the early stage, they may suggest lichen urticatus or papular eczema. In course of time, the skin becomes dark, rough, hard and thick, so that it cannot readily be pinched up between the finger and thumb. The affected parts may become the seat of eczema, and the neighbouring lymph glands may then undergo enlargement.

The disease is practically confined to the children of the poor. It is aggravated in cold and relieved in hot weather. In its worst forms it is incurable, though the slighter cases may ultimately recover under persevering treatment.

Treatment.—Relief is given by rest in bed, by alkaline baths, and by the inunction of carbolic oil. Cod-liver oil, iron and arsenic may be given internally; and pilocarpine may be injected hypodermically at bedtime, so as to cause sweating. Tarry preparations and other stimulating substances may be applied to the skin. The diet and hygienic conditions must, of course, have careful attention; and eczema, pediculosis, or any other cause of itching or urticaria, must be removed by appropriate measures.

7. Herpes.

Herpes Facialis (*H. febrilis*) is a localised eruption of vesicles on a red base. Its favourite seat is at the junction of the mucous and cutaneous surfaces of the lips, especially at the angle of the mouth. It is frequently bilateral, and may extend on to the cheek and involve the tongue. There may be one or several patches. The vesicles soon dry into dark crusts. Subjective phenomena are usually slight and may be absent. The affection is common in pneumonia

and in ordinary colds, but may have no obvious cause. It is liable to recur.

Treatment.—The affected part should be kept dry if possible, and may be dusted with zinc or starch powder. If it is impracticable to keep it dry, it should be dried gently and then anointed with zinc ointment or vaseline.

Herpes Progenitalis involves the prepuce, glans penis, labia, etc. If these parts are irritated by scratching or by the secretions, ulcers may result whose nature may be misinterpreted. The condition is apt to recur.

Treatment should be soothing.

Herpes Zoster (*zona*, *shingles*) is by far the most serious form of herpes. It is unilateral in distribution, and is often preceded, accompanied, or followed by severe neuralgic pain.

Etiology.—The disease occurs at all ages, but causes most suffering in the case of elderly subjects. It resembles the acute infections in beginning suddenly with fever (in well-marked cases), in manifesting a characteristic eruption at an interval after the onset, in running a definite course, in sometimes occurring in epidemic form, and in almost never recurring. In one of my cases, the blood yielded a culture of a diplococcus growing in chains. Pregnancy, phthisis, the internal use of arsenic, cold, and debility are among the causes of zoster, but in most instances no cause can be recognised.

Apart from the conditions just named, where zoster occurs spontaneously, the disease may be *symptomatic* of various organic affections of the nervous system, such as general paralysis of the insane, tabes dorsalis, meningitis, or pressure by a tumour upon a nerve or ganglion.

Morbid Anatomy.—There is acute and often hæmorrhagic inflammation of the posterior root-ganglion belonging to that segment of the cerebro-spinal axis from which the affected piece of skin gets its nerve-supply. Each fully-developed vesicle in the cutaneous eruption is a cavity containing serum and leucocytes, and also enormously swollen epithelial cells, whose nuclei multiply greatly so as to produce a kind of giant cell. The contents of the vesicle

usually become turbid and purulent, and the related lymph glands become large and perhaps tender.

Symptoms.—The principal early symptoms are shivering, slight fever, and pain in the region corresponding to the distribution of the nerve root involved.

The eruption appears after a varying interval, but generally after a few days. Erythema of the skin usually precedes the vesicles. The vesicles sometimes lead to ulcers, in which case scars remain. In other cases, a portion or the whole of the eruption aborts at the stage of papules, and never reaches the stage of vesiculation.

The pain varies much in intensity and duration. Old people in particular may suffer severely, and for a long time. Apart from the pain, the ordinary duration of the attack is from one to two weeks, but the disease may abort.

The eruption may appear almost anywhere, but the territories of the intercostal nerves, the lumbar nerves, and one or more branches of the trigeminal nerve are specially liable to suffer. In the supra-orbital form, the eye may become inflamed.

Diagnosis.—The pain and the vesicles are the leading symptoms. If the patient asks advice on account of the pain before the rash has appeared, the former is apt to be misinterpreted by the unwary. Zoster is distinguished from *herpes febrilis* by its unilateral distribution, by its situation, by its relation to a particular nerve territory, by the associated neuralgic pain, and by its non-liability to recur.

Prognosis.—The pain is occasionally very severe and persistent. In severe cases, considerable ulceration may take place and delay recovery, in addition to producing permanent scars. When the ophthalmic division of the fifth nerve is involved, the eye may be damaged.

A second attack does not occur in the same region as the first. In rare instances, a second and even a third attack may occur in different nerve territories.

Treatment.—The affected skin should be dusted with zinc powder or with boric acid and starch powder, and protected from irritation. Analgesics, such as acetanilide, phenacetin, phenazone, and in severe cases morphine, are

indicated. Fowler's solution (in 3-minim doses) has been recommended for the relief of pain in cases where arsenic was not the exciting cause.

8. Dermatitis Herpetiformis (Hydroa. Herpes Circinatus Bullosus. Pemphigus Pruriginosus).

This is a chronic, polymorphous skin disease characterised by recurring outbreaks of a widespread eruption.

Etiology.—This is obscure, but overwork and anxiety have been regarded as causes. The disease is most common in adults, though it may occur at any age. It sometimes appears during or after pregnancy, and after passing away altogether or partially, reappears or becomes aggravated with later pregnancies (*Herpes gestationis*). One theory is that it is due to toxæmia ; another that it is a neurosis.

Symptoms.—The principal elementary lesions are erythema, vesicles, and bullæ, the two latter tending to arrange themselves on or around the former. Papules, macules and pustules may also occur. The lesions group themselves into rings and small patches. They may involve the mucous membranes (*e.g.*, of the mouth) as well as the skin. The individual eruptions appear somewhat suddenly, and there is simultaneous aggravation of the itching, which is generally an important symptom. Pigmentation and infiltration of the skin are common. Individual lesions may last for a period varying from hours to weeks, but repeated crops of lesions develop. Eosinophile cells are generally present in the blood in unusual numbers. They are also found in the lesions along with other kinds of cells. In spite of the extent of the lesions, and the amount of discomfort (including itching of the skin, and burning and soreness of the mucous membranes) suffered by the patient, the general health is but little affected in most cases.

Diagnosis.—The most important features are the herpetic type of eruption (vesicles and bullæ), the itching at the outset of each crop, the polymorphous character (various kinds of lesions occurring at the same time or following one

another quickly or slowly), and the recurrence at irregular intervals and during an indefinite period.

Pemphigus is associated with more marked constitutional symptoms, but with less itching. The bullæ are usually larger than in dermatitis herpetiformis, and moreover they develop on previously healthy skin ; whereas in dermatitis herpetiformis, the vesicles appear on an erythematous patch. Pemphigus is not so definitely polymorphous.

In *erythema multiforme*, itching is slight or absent, the duration is limited, and vesicles and bullæ are not common.

Prognosis.—The disease lasts for months or years, and is to be regarded as a chronic affection with many recurrences, relapses or recrudescences. It may ultimately pass off spontaneously, but is occasionally fatal.

Treatment.—Rest, plain diet and good hygienic conditions are important. In some cases arsenic is of service, while in others it does harm. Sedatives may be necessary, such as phenazone or opium. Among external remedies, sulphur ointment, calamine lotion, and oils containing tar, ichthyol, thymol or carbolic acid may be mentioned.

9. Pemphigus.

This is a disease whose principal characteristic is an eruption of bullæ, but as these occur also in erysipelas, erythema multiforme, dermatitis herpetiformis, purpura rheumatica, etc., their presence is not pathognomonic.

Pemphigus Vulgaris (*P. chronicus*) is characterised by the development of vesicles which grow into bullæ. The skin at the seat of lesion is at first healthy looking, but later on a pink zone may surround the blebs. The bullæ may be scarcely bigger than vesicles, or may be as large as a hen's egg. They often measure ½ to 1 inch in diameter. Their number varies greatly. They contain serum which at first is clear, but afterwards becomes turbid. They are tense at first, but in the course of some days they collapse, owing to absorption of the fluid, or to rupture. After the crusts fall off, stains may persist for a time, but there is no scarring. Successive crops and relapses may prolong the disease for weeks or months.

Prognosis.—Childhood, advanced years, numerous relapses, great abundance of the lesions and the development of fever render the prognosis grave, and death may result. On the other hand, short attacks in adults do not necessarily affect the general health, and may end within a few months in perfect recovery.

Pemphigus Foliaceus is very rare, and is one of the few diseases (the others being *Pityriasis rubra* or exfoliative dermatitis, and *Lichen ruber*) which may involve the entire cutaneous surface. It often begins like chronic pemphigus, but the bullæ are flaccid from the first. In some cases a single bulla develops on the chest, and while drying at its central part into a thin yellow crust, spreads at the periphery, until in the course of perhaps many months the whole of the body is involved. As the crusts become detached, they expose a raw surface, which remains moist, and continues to pour out a semipurulent and often very offensive discharge.

Diagnosis.—The yellow crusts, the patches of raw surface, and the offensive secretion might suggest *eczema*, but the latter is never universal.

Prognosis.—The constant discharge from the universally affected surface, and the pain, loss of sleep, septic absorption and fever almost always lead to a fatal issue, though there may be periods of remission with marked temporary improvement.

Pemphigus Vegetans (*Erythema bullosum vegetans*) is a rare disease which involves the regions of the flexures. After the blebs have been replaced by crusts, condylomatous-looking growths or vegetations develop. The disease is almost always fatal.

Pemphigus Acutus is less common than the chronic form. It attacks children more frequently than adults, and commences with severe constitutional symptoms, including fever. The bullæ develop in successive crops, and are very apt to rupture. The mucous membranes may suffer as well as the skin. Many cases end fatally, owing in great measure to septic absorption. A specially severe type (*P. acutus malignus*) is met with in butchers, tanners and others who handle animal matter. It appears to be due to infection

by a special diplococcus which has been found in the contents of the bullæ.

Pemphigus Acutus Neonatorum is characterised by the development of bullæ in newly-born children who are not properly cared for. The lesions may be scattered over the whole of the trunk and limbs. The disease is doubtless a result of infection by pyogenic microbes, and a staphylococcus and streptococcus have been found. There is little or no constitutional disturbance, and recovery is the rule. It must be distinguished from the bullous eruption ('syphilitic pemphigus') occasionally observed in syphilitic infants, especially on the palms and soles.

Treatment.—In *pemphigus vulgaris*, arsenic may be tried in large doses, but there is now some doubt whether it actually merits the high reputation it formerly enjoyed. Quinine and opium may also be tried. Dusting powders mixed with boric acid, camphor, or menthol, should be applied locally. In *pemphigus foliaceus* arsenic is of little use, and a continuous medicated bath at a suitable temperature is indicated. In all cases of pemphigus, good food, clothing and air are important.

10. Pityriasis Rubra (Dermatitis Exfoliativa).

This is a chronic or acute, general or universal, non-contagious dermatitis, characterised by intense redness and profuse desquamation.

Etiology.—The affection is most common in adults. Gout, rheumatism, kidney disease and chronic alcoholism are predisposing causes. The disease may be primary or secondary. The *primary* form is frequently due to chill, and occasionally to mercurial poisoning; whilst the *secondary* form develops in connection with psoriasis, eczema or some other cutaneous ailment. *Dermatitis exfoliativa neonatorum* (*Ritter's disease*) is perhaps a variety of this affection.

Symptoms.—The onset may be acute with shivering and fever, or it may be gradual. The eruption commences in the form of one or more patches which spread till the whole or a great part of the body is involved. The colour is deep red or even livid. The eruption is a dry one, but there may

be fissures (*e.g.*, at the flexures) from which oozing takes place. Desquamation is excessive, and pints of scales may be thrown off in a day. The scales vary in size : some are small and branny, and some are large and membranous. Itching and infiltration are not prominent features, though there may be tenderness and a feeling of tension. The nails frequently become soft, and may be ultimately shed. The mucous membranes are sometimes involved. The patient may be very sensitive to cold.

Diagnosis.—This should seldom be difficult. The history of the case, the universal distribution of the eruption, the marked redness and dryness of the skin, and the profuse desquamation are sufficiently characteristic.

Pemphigus foliaceus may give rise to a universal eruption, but here there are bullæ and an offensive discharge from the skin.

Lichen ruber (*L. planus*) is a third disease which may involve the entire cutaneous surface, but here the eruption consists of papules.

In *psoriasis*, the scales are firmly adherent to the affected surface, are silvery in appearance, and are not aggregated into large membranous flakes. The eruption shows a preference for the extensor aspect of the knees and elbows, and is rarely universal.

Prognosis.—Recrudescences and relapses are common. Most cases recover spontaneously after many months, but in some instances albuminuria, diarrhœa, and other complications lead to a fatal issue. Ritter's type is particularly dangerous.

Treatment.—Rest in bed between blankets, abundance of light nourishing food, and diuretics, are indicated at the commencement. Cod-liver oil, strychnine, and quinine should be given as supporting agents or tonics. Soothing applications such as diachylon ointment, Lassar's paste,[1]

[1] Zinc oxide and powdered starch, of each ʒii., vaseline ʒss, salicylic acid gr. x. The paste should be spread on thickly, and covered with butter cloth. It may be cleaned off with olive oil. If the inflammation is acute, the salicylic acid should be omitted, or replaced by a milder antiseptic such as ichthyol.

linimentum calcis, or pure vaseline, should be used locally. If there is itching, resort should be had to warm bran baths (6 pounds bran to 30 gallons of water).

11. Dermatitis Exfoliativa Epidemica.

In its appearance, this disease is the same as the non-contagious pityriasis rubra. It is, however, contagious and microbic, and in recent years it has repeatedly come under notice in epidemic form in institutions.

The microbe which is constantly present in the skin in this disease is a diplococcus. It has also been found in the blood and urine, and in pus from superficial pustules, as well as in the bedclothes. It has been found after death in the spleen, in subcutaneous abscesses, and in pyæmic abscesses in the lungs and kidneys.

Albuminuria, diarrhœa, and boils are among the complications.

Prognosis.—In old and feeble subjects, there may be fatal prostration.

Treatment.—Good feeding and antiseptic ointments constitute the main part of the treatment.

12. Pityriasis Rubra Pilaris.

The characteristic lesions begin on the hairy parts of the limbs or body in the form of reddish papules which give the skin a rough feeling. They send down processes into the hair-follicles, so that when they are rubbed off, little pits are left. The hairs are strangled and damaged. The lesions generally persist in this form throughout the disease, on the backs of the fingers and toes ; but in other parts, for instance about the elbows and knees, they become confluent and give rise to patches very like those of psoriasis. The skin between the patches becomes red and desquamates, the palms and soles are scaly, and seborrhœa is well marked on the head.

Some authors consider this disease to be the same as *Lichen ruber acuminatus*.

Recovery ultimately takes place as a rule, but a relapse

may occur, and the disease is occasionally fatal. Diaphoretics and thyroid extract may be given internally; and ointments of tar or salicylic acid may be applied to the skin.

13. Lichen Ruber.

Two principal varieties of this disease are recognised, the one being localised and the other generalised.

Localised Lichen Ruber (*L. ruber planus, L. planus*) is not very rare in Britain and America. It may occur at any age, but is seldom met with in children. With the exception of nervous exhaustion from overwork, anxiety, etc., it is not possible, as a rule, to assign any cause for the attack.

The eruption consists of papules which are at first small, but may grow to some size. They are purple or red in colour, angular in shape, smooth on the top, and either flat-topped or umbilicated owing to the presence of a follicle. The papules, as they increase in number and size, may become confluent, so that patches arise which are surrounded by isolated papules. The rash is always dry, and on fading, it leaves dark stains, but never scars. Itching is seldom severe.

The eruption, which is usually symmetrical, may attack any part of the body, but its favourite seats are the anterior aspect of the wrists and forearms, and the legs. It sometimes involves the mucous membrane of the mouth.

The course is usually chronic (*chronic lichen planus*), but ultimate recovery is the rule.

A minority of cases of this disease run a more rapid course (*acute lichen planus*). The eruption spreads rapidly over the whole body, and at first there is a diffuse erythema in addition to the papules. The erythema soon passes away, and the papules in their turn are absorbed, leaving stains to mark their places. The duration of such an attack is perhaps a few months, and the subjective symptoms may be very slight indeed.

Generalised Lichen Ruber (*L. ruber acuminatus*) appears to be more common in Austria than in Britain or America. The disease may begin in the same way as

L. planus, but the papules are conical instead of flattened, and are crowned with fine, white scales. The papules increase rapidly in number and become confluent, until considerable areas or even the whole surface of the skin may be involved. In this way the whole skin may be reddened, thickened and scaly. The thickening is specially marked in the palms and soles. Fissures develop and may bleed, and the movement of joints is interfered with. Changes take place in the nails.

In cases where the disease involves much or the whole of the cutaneous surface, the general health is apt to deteriorate, and after a very prolonged illness the patient may die of exhaustion.

It is to be noted that good authorities are disposed to separate *L. acuminatus* altogether from *L. ruber*, and to regard it as identical with *Pityriasis rubra pilaris* (p. 913).

Treatment.—Attention should be given to the general health, and good food, rest of body and mind, tonics and a change of air may be prescribed. Opium or some other sedative may be necessary. Arsenic and mercury should have a thorough trial as internal remedies, after any constitutional disturbance has been rectified. By way of local treatment, soothing measures should be employed in acute cases with erythema ; whilst in the chronic varieties, stimulating remedies such as tarry preparations are indicated.

The term LICHEN was formerly applied to a large number of cutaneous diseases whose common feature was that the elementary lesion was a papule. Many of these affections have since been detached from the group of lichens and classified according to their true affinities. Thus *L. simplex* or *L. infantum* (strophulus, erythema strophulus, stomach rash, red gum) is to be regarded as a papular eczema. *L. agrius* or *L. eczematosus* (eczema lichenoides) is also papular eczema. *L. urticatus* is urticaria papulosa. *L. circumscriptus* or *L. circinatus* is probably either seborrhœic eczema or seborrhœa corporis. *L. tropicus* (prickly heat) is miliaria papulosa. *L. pilaris* is not inflammatory, and is better named keratosis pilaris. *L. scrofulosus* has been described among tubercular affections (p. 139).

14. Acne (Acne Vulgaris. Acne Disseminata).

Acne is a chronic affection of the skin which is chiefly seen on the face, shoulders and upper part of the chest. It is an inflammation of the sebaceous glands and hair-follicles which results from retention of the secretion. Acne is frequently associated with comedones, in which there is retention without inflammation (p. 888).

The disease is most common in the period from fifteen to twenty-five years of age, and it has been supposed to be related to the development of the sexual function. The eruption consists of pimples, which appear in successive crops, and after suppurating leave minute scars. Sometimes there is a good deal of induration around the pimple (*A. indurata*). In severe cases, one or more of the sebaceous glands may become the seat of small boils, which suppurate slowly, and leave scars like the pits of small-pox.

Diagnosis.—This is generally easy. Syphilitic eruptions are distinguished by not being confined to the face and chest, by the absence of comedones, and possibly by the age of onset, the history, and other evidences of syphilis. It must be remembered that the internal use of bromides and iodides may give rise to acne.

Treatment.—Comedones must be removed. The face should be thoroughly scrubbed each night with soap and hot water, and a sulphur lotion or ointment may be applied thereafter. If the scrubbing causes much irritation of the skin, a calamine and zinc oxide lotion may be applied for the first few nights ; the lotion is allowed to dry on the skin, and the latter is washed with plain water in the morning. Cod-liver oil and arsenic may be given internally. Obstinate cases of acne may be treated by Wright's plan of inoculating a staphylococcus vaccine of standard strength. The use of the vaccine is regulated by systematic examinations of the phagocytic or opsonic index of the blood.

15. Acne Rosacea (Rosacea. Gutta Rosea).

Rosacea is quite distinct from acne, though the two may occur together. It is much more common in females than

in males. Menstrual disturbance and dyspepsia in women, and alcoholism in men are important causes. In males it tends to begin in middle life ; in females it begins especially at puberty or at the menopause.

The parts affected are the nose, cheeks, chin and forehead. There is redness of the skin with dilatation of vessels and an oily secretion (*seborrhœa nasi*). Papules and pustules may develop. The congestion is apt to be aggravated after alcoholic or hot drinks, and to be then accompanied by a burning sensation.

In the *hypertrophic* variety (*rhinophyma*), which is seen chiefly in men, and is sometimes though not always due to alcoholic excess, there is great overgrowth of the soft tissues of the nose.

Treatment.—It is important to correct any uterine or digestive disorder, and to put a stop to excesses in alcohol, tea, etc. In ordinary cases alcohol should be forbidden, but if there is debility, tonics may be beneficial. Dilated vessels may be slit open longitudinally, and after the wounds have healed, a mild sulphur ointment may be rubbed in each evening. In the morning, the face should be washed with oatmeal-water or gruel, and after drying, a little calamine and zinc oxide lotion should be allowed to dry upon it. Electrolysis, however, is a better method than incision for the ordinary form. In cases characterised by hypertrophy without definite tumour, or by a great aggregation of pustules, multiple scarification should be employed.

Definite tumours require surgical measures.

16. Ecthyma.

This term is applied to the condition in which large and not very numerous discrete pustules and scabs are found scattered over the limbs and trunk in debilitated subjects. It may be looked upon as a phase or result of impetigo contagiosa, and is associated, like the latter, with the presence of the streptococcus.

Treatment.—Tonics, good food and hygiene, removal of crusts, and applications of white precipitate ointment constitute the appropriate treatment.

17. Drug Eruptions (Dermatitis Medicamentosa. Toxicodermiæ. Medicinal Rashes).

The administration of certain drugs is liable to give rise to skin eruptions, the more frequent varieties of which must be borne in mind by the practitioner. Individual peculiarity is very influential.

Acetanilide (antifebrin).—A slate colour suggestive of cyanosis, but probably a dyeing effect.

Antitoxin (diphtheritic).—Erythema and urticaria.

Arsenic.—Erythema, urticaria, eczema and zoster. Prolonged use may cause pigmentation of the skin and thickening of the epidermis (keratosis) of the palms and soles.

Belladonna.—A scarlatiniform rash.

Borax.—A scaly rash like psoriasis.

Bromides.—Acne often ; sometimes erythematous, urticarious and other eruptions.

Cinchona and *Quinine.*—A scarlatiniform, measly or urticarious eruption.

Chloral hydrate.—Erythema, urticaria, and other eruptions.

Copaiba and *Cubebs.*—Bright red, itchy patches which may spread over most of the body.

Digitalis.—Scarlatiniform, papular, and urticarious rashes.

Enemata.—If made with ordinary hard soap, large enemata not uncommonly cause a scarlatiniform rash which may after a time become urticarious. The use of soft soap does not appear to cause a rash. I have seen a similar eruption follow the accidental swallowing of a piece of hard soap.

Iodine and *Iodides.*—Acne-like, erythematous, bullous and purpuric eruptions.

Iodoform.—Erythema, urticaria, and purpura after absorption ; a vesicular eruption locally.

Opium and *Morphine.*—Sudamina, erythema, and urticaria.

Phenazone (antipyrin).—An erythematous or measly rash ; sometimes papular, urticarious, purpuric, etc.

Salicylates.—Scarlatiniform, measly, urticarious and other eruptions.

Santonin.—Urticaria.

Sulphonal.—Scarlatiniform and other rashes; purpura (with hæmatoporphyrinuria).

Tar.—Erythema.

Turpentine.—Erythema.

Treatment.—Sometimes the medicine must be withdrawn for the time, or reduced in amount, or avoided altogether for the future; but in certain cases, as in the prolonged administration of bromides or of borax in epilepsy, the eruption may be completely prevented by the addition to each dose of a small quantity of arsenic.

18. Psoriasis.[1]

Psoriasis is a very common and important disease of the skin, and is characterised by overgrowth affecting specially the deeper layers of the epidermis, with the result that patches of dry, silvery scales develop on a red base.

Etiology.—Hereditary tendency seems to be the only undoubted cause which can be recognised. Those who suffer from the disease are specially liable to fresh attacks in winter and in spring. To such individuals, moreover, sea-air and sea-water are frequently hurtful. In those who are liable, an attack may be induced by debility. Thus, women are apt to suffer during lactation, and McCall Anderson quotes a case where the patient suffered only while suckling her male children. I have met with a case where the disease occurred after the births of the last four children, who were all girls, but not after that of the first child, who was a boy. In this patient, moreover, the eruption affected that part of the left ring-finger which was in contact with the ring. By way of experiment she transferred the ring to the right ring-finger, with the result that the disease appeared there also. The fingers were unaffected except where they were in contact with the ring. Local irritation, however, by itself, is very rarely a cause of psoriasis.

Psoriasis is rare in the first three years of life, but is common in older children and young adults. Most cases

[1] ψώρα = scurf; formerly called *lepra*.

begin before the thirtieth year. Both sexes and all classes of society suffer practically alike.

Symptoms.—The lesions begin as minute papules, which are crowned with scales almost from the first. The silvery character of the scales is attributable to the presence of air among them. If it is not well marked, it can be brought out by scratching. The patches are distinctly demarcated from the surrounding healthy skin. In the early stage they are very small (*Psoriasis punctata*), but they grow to be ½ an inch or 1 inch in diameter, and then resemble drops of mortar (*P. guttata*). These spots often coalesce into considerable patches. The scales are very adherent, and when they are torn off, slight bleeding may result. Sometimes the scales are heaped up to such an extent that the patch, as in rupia (see p. 146), resembles a limpet-shell (*P. rupioides*).

The tips of the elbows, and the knees just below the patellæ are almost always affected. The head also suffers frequently, though the face generally escapes. Almost any part of the body, however, may be involved. Sometimes the disease attacks the nails, causing them to become thick, opaque, and brittle. The distribution is roughly symmetrical, and as a general rule, the eruption is more abundant on the back than on the front of the trunk, and on the extensor than on the flexor aspect of the limbs. The eruption is dry, and itching is usually slight, unless in gouty subjects. The general health is often very good.

Diagnosis.—A *squamous syphilide* (syphilitic psoriasis) is sometimes difficult to distinguish from psoriasis. There may be other signs of syphilis, a history of syphilitic infection and marked coppery pigmentation ; the scales are not so silvery as in psoriasis, the elbows and knees are not so constantly affected, and the palms and soles are frequently involved. On the other hand, in simple psoriasis the palms and soles are seldom involved, while the knees and elbows almost always are. The fact of the eruption having been present at an early age points to simple psoriasis. Simple psoriasis is apt to relapse repeatedly during a long period of years ; syphilitic psoriasis, once cured, is not so likely to recur.

Mercury and potassium iodide will cure the syphilide; arsenic will often cure psoriasis.

In *eczema squamosum* there are often patches of eruption which are or have been leeting; the scales are seldom silvery; itching is present; and the extensor aspects of the elbows and knees are not likely to be involved.

In *dermatitis exfoliativa* (*pityriasis rubra*) the skin is deep-red all over, and not simply in spots or patches; the epidermis peels off in large membranous flakes; and the scales are not situated on a raised base.

Ichthyosis is present from infancy; the scales are brown or dirty-looking; the skin is dry all over; and there are no signs of inflammation.

Seborrhœa of the scalp forms greasy crusts over pale skin.

Prognosis.—This is favourable as regards life, but relapses occur in the great majority of cases. It is often difficult to remove an attack, but spontaneous disappearance is sometimes observed.

Treatment.—Arsenic is the most important remedy. Fowler's solution or the acid solution should be given after meals in doses which may be small at first but should be gradually increased almost to the limit of tolerance for the particular individual. Arsenic should not be given, however, while the eruption is coming out acutely.

Other internal remedies are carbolic acid (gr. ii.-iv. thrice daily in pill), creosote (♏. xv.), ammonium carbonate (gr. x.-xxx.), potassium iodide (large doses), salicin (gr. xv.), sodium salicylate, cod-liver oil, and—in a patient who can be kept under constant supervision in bed—thyroid extract. Any source of debility must of course be removed.

The treatment should be local as well as general. The patient should have alkaline baths once or twice a day, so that the scales may be removed by scrubbing with a nail-brush. Thereafter some stimulating ointment, liniment, or lotion is to be applied. The milder preparations are mercurials, such as white precipitate ointment. Stronger than these are tarry substances, such as ointments or liniments of oil of cade or of birch-tar (♏x.-℥ii. in ℥i.). Another remedy is liquor picis carbonis (℥ss.-℥ii. in ℥ii. of water).

One of the strongest and most useful preparations is chrysarobin, which may be used as an ointment in the strength of gr. x. in ʒi. It is rubbed into the affected parts (excluding the head) night and morning. As it stains the linen, old clothing must be used during the course of treatment. Sometimes it causes erythema of the skin.

In acute cases, the treatment just described is contra-indicated; the applications must at first be soothing and not stimulating.

19. Lupus Erythematosus (Lupus Erythematodes. Batswing Lupus. Butterfly Lupus. Ulerythema Centrifugum).

This is a peculiar inflammatory eruption which in the great majority of cases is confined to the nose, cheeks and ears, though occasionally involving the hands and other parts, either alone or in combination. The distribution of the disease on the nose and cheeks suggests the form of a bat or butterfly. The affection is one of adults, but it tends to die out in old age. It is much more common in women than in men. Its nature is still uncertain. It has been regarded as tubercular, but the correctness of this view has not yet been demonstrated. Lupus erythematosus is specially apt to occur in persons with a feeble circulation, including those who complain of chilblains; and also in tubercular and other debilitated subjects. It is not contagious, auto-inoculable, or hereditary.

Morbid Anatomy.—The epidermis is very thin. The underlying corium is œdematous and infiltrated with leucocytes and connective-tissue cells. The condition is a chronic inflammation beginning at the bloodvessels, and associated with dilatation of lymph channels and sometimes with thrombosis. Secondary atrophy ensues. The inflammation sometimes attacks specially the regions of the glands and hair-follicles.

Symptoms.—The disease may be seen in an early stage in the form of little red spots which afterwards increase and coalesce to form rounded patches. The original spots are

covered by scales, and when a scale is removed, it is found to have adherent to its under surface an epidermic plug which had projected into a sebaceous follicle or some other depression in the epidermis. The patches are elevated at the margins, and while they spread at their periphery, they may heal at their centres, giving rise to a slightly depressed scar. There is no ulceration. The disease may cause itching or burning, but seldom actual pain. In rare cases it becomes generalised over almost all the body (*L. erythematosus disseminatus vel exanthematicus*).

Diagnosis.—*Lupus vulgaris* generally begins before adult life. Isolated, apple-jellylike nodules may be recognisable. Ulceration is common. The sebaceous glands are not specially involved. Symmetry is not a feature.

Chilblains do not persist through summer, and do not show cicatricial atrophy of the skin.

In *seborrhœa sicca*, the crusts consist largely of sebaceous matter. The skin beneath is pale or but slightly reddened, and is oily. There is no scarring.

Prognosis.—The disease shows but little inclination to disappear, and may persist for an indefinite number of years. It is most refractory to treatment. Some cases, however, undergo cure pretty quickly ; while, on the other hand, the rare disseminated form may prove fatal.

Treatment.—Cod-liver oil, arsenic and other tonics may be given internally. Salicin, quinine, and ichthyol are other drugs which have been recommended. Soothing applications such as calamine lotion are indicated in the active stage, and under such measures, the disease may become inconspicuous. In the quiescent stage, the part may be rubbed daily with flannel and soap spirit till all the scales are removed, or collodion containing 10 per cent. of ichthyol or of soft-soap may be applied. In all cases the affected parts should be protected from abrupt or extreme variations of temperature.

20. Cheiropompholyx[1] (Pompholyx. Dysidrosis).

This disease manifests itself as a symmetrical eruption of vesicles on the hands, especially along the borders of the fingers. It sometimes occurs also on the feet. The vesicles have a transparent appearance, like grains of boiled sago. They are well embedded in the skin, and have little or no tendency to rupture. In the course of some days they dry in, to be afterwards exfoliated. The attack passes off within a few weeks.

Apart from the vesicles, and some heat and itching, there may be few or no signs of inflammation, but eczema and other inflammations of the skin may follow.

Etiology.—Those who perspire readily are specially liable to this disease, but there is reason to believe that the vesicles are inflammatory in their origin and are not due merely to retention of sweat. Local irritation and a congenital tendency are mentioned as causes, in addition to hyperidrosis. The affection may occur at any age.

Prognosis.—The slighter attacks pass off after days or weeks, but sometimes the disease assumes a chronic phase ; and though recovery at last takes place, there is a strong tendency towards recurrence.

Treatment.—Tonics may be given internally. A dusting powder or calamine lotion may be applied locally. The affected parts should be kept thoroughly clean.

21. Sycosis (Coccogenic Sycosis. Pustular Folliculitis of the Beard.[2] Mentagra).

This disease is generally, though not by all dermatologists, recognised as distinct from eczema of the hairy part of the face. It is an inflammation set up, in or around the hair-follicles, by pyogenic micrococci. Acne-like papules appear and develop into pustules, through the centre of each of which a hair passes. In acute cases there may be con-

[1] χείρ = hand ; πομφόλυξ = bubble.

[2] A somewhat similar *pustular folliculitis of the scalp* is known as *Quinquaud's disease.*

siderable infiltration, but the disease is usually very chronic and spreads slowly.

Diagnosis. — The distinction from *eczema* may be very difficult. In eczema there is inflammation of the skin, to which pustules round the hair-follicles may be added ; in sycosis, the pustules at the hair-follicles are the essential feature, though the intervening skin is apt to become inflamed. In sycosis the pustules are likely to be more numerous than in eczema. Sycosis occurs chiefly in adult males ; it is attended by pain or burning, but there is little or no itching, and it is confined to the hairy parts.

Impetigo is not likely to be limited to the hairy parts. Moreover, it develops more quickly, and produces more discharge than sycosis.

Ringworm of the beard generally begins with a circular patch, and is usually associated with hard, deep-seated nodules. It spreads more quickly than sycosis, and is associated with a fungus.

Treatment. — Crusts should be moistened with oil, fomented, and removed. Thereupon the hair in the affected region should be cut short, and every hair which is seen to pass through a pustule should be removed by epilating forceps, after which white precipitate ointment should be well rubbed in. At a later period, it is well to epilate the whole of the affected area, a small proportion of the hairs being removed daily. A parasiticide ointment should be rubbed in after each operation ; or, from the first, epilation may be carried out by the agency of the X rays, and followed by the application of the parasiticide ointment.

ii. New Formations.

1. Keratoses.

(1) *Clavus* (*Corn*).

This condition is most common on the foot because boots are so often tight or ill-fitting. The visible part of the corn is an overgrowth of epidermis which projects above the level of the skin. The deeper part is an inverted cone—the 'root' or 'eye' of the corn—and likewise consists of

epidermis. Its apex presses inwards upon the sensitive corium. The corium is thus depressed and irritated, and may become inflamed and atrophied. Pressure on the corn naturally causes pain, but spontaneous pain is often induced by atmospheric conditions. An old corn comes to resemble horn in colour and hardness, but a corn situated between the toes, where the skin is always more or less moist, remains white and soft (*soft corn*). Occasionally a bursa develops underneath the corn, constituting a *bunion*, and this may become the seat of inflammation and suppuration.

Treatment.—A convenient palliative measure is bathing the foot in hot water, and then paring the corn with a razor. 'Corn-cures' generally depend upon salicylic acid, which destroys the morbid epithelium while sparing the healthy tissues. Salicylic plaster may be applied daily for some days and kept in place by a bandage; or a solution consisting of salicylic acid ʒi. and extract of cannabis indica gr. x. in flexile collodion ʒi. may be used each evening. After some days, when the plaster is removed, the latter may pull off the corn with it, or the corn may be scraped away.

Attention should be given to the shape and size of the boots.

(2) *Callosity* (*Tylosis. Tyloma*).

This is a simple thickening of the epidermis, often of considerable size, but not associated with any core pressing downwards upon the corium, and therefore not attended with pain. It is chiefly seen in the hands and feet. In the case of the hands, it is often the result of the patient's occupation. Great overgrowth of the horny epidermis of the palms and soles occurs as a rare disease which may run in families through several generations (hereditary keratoma or tylosis).

(3) *Cutaneous Horn.*

Cutaneous horns are rare in the human subject. They are most common on the head, but may occur almost anywhere, and may reach a length of many inches.

The **treatment** consists in removal of the horn, and cauterisation or excision of the cutis from which it grows.

(4) *Keratosis Pilaris* (*Lichen Pilaris*).

Keratosis pilaris is not an inflammatory disease, though scratching may occasionally give rise to some congestion. It takes the form of little papules about the size of pins' heads, which develop around the hairs of the legs, thighs and arms. The papules consist of débris which accumulates inside the follicles and projects from them.

Treatment.—This consists in thorough scrubbing with soap and hot water, with subsequent inunction of glycerin.

(5) *Verruca.*

Verruca Vulgaris (*common wart*) occurs most commonly on the hands. It is due to hypertrophy of a small group of papillæ, and may be smooth or rough ; dark, or coloured like the surrounding skin. It is generally horny on the surface and firm.

Treatment.—To remove a wart, the epidermis may be pared away, and the hypertrophied papillæ may then be cauterised with glacial acetic acid, carbolic acid, or nitric acid. Or a saturated solution of caustic potash may be employed. Care must be taken that the surrounding skin is protected from the caustic. For some warts, nothing is better than excision with a pair of scissors.

Verruca Plana (*flat wart*) is seen in elderly people (*verruca senilis*), especially about the face, neck and back. It is dark in colour, and is but little elevated, though more extensive in area than the common wart.

Verruca Acuminata (*cauliflower excrescence*) has a special tendency to develop on the glans penis, on the under surface of the prepuce and on the inner surface of the labia. It often accompanies venereal affections in consequence of the irritation of discharges (*venereal wart*), but it may occur in those who have never had venereal disease, and in other moist portions of the skin besides the genitals. As it is

often moist, it is sometimes spoken of as *moist wart*. It appears in the form of groups of pointed elevations, which may be either sessile or pedunculated, and have frequently a pink or red colour. It is important not to mistake them for condylomata.

In the **treatment** of verruca acuminata, cleanliness should be secured by frequent washing with a weak solution of potassium permanganate, after which a dusting powder should be applied.

Verruca Digitata occurs on the scalp. It gives rise to a broad mass in which the hypertrophied papillæ are not blended together by thickened cuticle, but project as finger-like processes.

Verruca Filiformis is a thread-like projection due to hypertrophy of one or a few papillæ. It occurs on the eyelids, neck and elsewhere.

Verruca Necrogenica (*cadaveric wart, anatomical tubercle*) is a tubercular lesion (see p. 139).

(6) *Ichthyosis* (*Fish-skin Disease*).

Ichthyosis is a congenital abnormality of the skin characterised by hypertrophy of the papillary layers and hardness and brittleness of the epidermis. The latter cracks along the lines of the skin, so that lozenge-shaped scales are produced. The condition is met with in many degrees of severity. Slight cases are known as *xerodermia*, and are characterised by abnormal dryness and roughness of the skin, from various parts of which—*e.g.*, on the sides of the chest—fine, tough, adherent scales project. The skin looks dirty, and though it may appear to be cleaned by very thorough washing, a few hours may suffice for a restoration of the dirty aspect. In more marked cases (*I. simplex*) the scales or plates, which are free at their edges but adherent at their centre, and which are separated from one another by distinct fissures, strongly suggest the skin of a reptile (*I. serpentina*). In exceptional cases, the plates project like warts (*I. hystrix*), and individuals thus disfigured have exhibited themselves as ' porcupine men.'

Ichthyosis commonly escapes notice during the first six months of life. It is often inherited and runs in families. It attacks both sexes, though often confining itself to one sex in a family. The general health does not suffer.

Treatment.—Considerable improvement may be effected by treatment. The general condition may be improved by cod-liver oil, arsenic and iron. The less adherent epidermic scales should be removed by alkaline baths, and friction with soft soap and hot water. Hypodermic injections of pilocarpine, and the cautious administration of thyroid extract may be tried, in order to promote sweating and the removal of crusts. The skin should be rubbed with lanolin. In severe cases it is desirable that the patient should reside in a warm and moist climate.

2. Scleroderma.

Diffuse Scleroderma (*sclerema, scleriasis, dermatosclerosis, hidebound disease*) is a chronic disease characterised by an overgrowth of fibrous tissue in the skin, which causes the latter to become hard and immobile. The fibrous growth involves not only the corium, but also the subcutaneous fatty tissue, deep fasciæ and periosteum. In the long-run, the fat disappears, and the true skin and the glands are atrophied.

The disease is more common in women than in men. It is most frequent in early adult life, and at middle age. It is often unilateral. The upper half of the body and the upper limbs suffer most. When the fingers are specially involved, the condition is sometimes known as *sclerodactylia*. The onset is very gradual. The skin becomes hard, so that it cannot be pinched between the finger and thumb. Voluntary movement is interfered with. The surface is generally smooth and shiny, and may be pigmented.

In a few cases the mucous membrane of the mouth is affected. The sensory functions are not much if at all disturbed. Raynaud's disease is sometimes associated with sclerodermia.[1]

[1] See the author's monograph on 'Raynaud's Disease,' p. 163 *et seq.*

Prognosis.—Spontaneous cure may take place, but if the disease progresses for a long time, the patient may become debilitated, and he is thus rendered an easy victim to intercurrent disease.

Treatment.—Tonics, cod-liver oil, change of air and shampooing are the measures indicated. Salicin, salicylates, and occasional Turkish baths also deserve a trial.

Circumscribed Scleroderma (*morphœa, Addison's keloid*) usually occurs as a rounded or oval patch, but sometimes it follows the course of a superficial nerve. At first it may be pink in colour, but later on it becomes yellowish-white, smooth, and firm like ivory. The patch is well defined, being surrounded by a coloured areola due to dilated vessels. The neighbouring skin may become pigmented. The patches may be single or multiple, and are sometimes the seat of itching or pain.

Scleroderma Neonatorum (*sclerema neonatorum*) is observed at birth or within a few days thereafter. It usually begins in the legs and tends to spread upwards till a great part or the whole of the body is involved. The skin becomes cold, hard, and pale or mottled. Pressure with the finger suggests that the skin is half frozen. The limbs and face are immobile. Death generally results after days or weeks from inanition or some complication.

Treatment.—The child should be kept warm in an incubator, or by wrapping it in cotton wadding. The body should be rubbed with warm oil, and abundance of warm milk should be given.

3. Xerodermia Pigmentosa (Atrophodermia Pigmentosa. Xerodermia Maligna. Lentigo Maligna Juvenilis. Kaposi's Disease).

This is a very rare disease, which generally commences in the first or second year of life, and tends to run in families, though not to be inherited. Six kinds of lesion can usually be recognised :

1. Pigment spots exactly resembling freckles (lentigo), both in their appearance and in their tendency to involve chiefly the uncovered parts.

2. Telangiectases, or dilated vessels, which appear, after months or years, between the pigment spots.

3. Small, white, atrophic areas scattered among the pigment spots on the face.

4. Warts of various sizes, many of them resembling senile warts.

5. Superficial ulcerations with yellowish or greenish crusts, situated chiefly on the face, and tending as they heal to produce cicatricial deformities.

6. Tumours, at first papillomatous but ultimately epitheliomatous, taking origin in the warts and ulcers, and leading to a fatal issue unless they can be removed in time.

Conjunctivitis and pterygium are almost constantly present.

The prognosis is bad, but by appropriate treatment of the ulcers, and by removal of the tumours, the epitheliomatous stage may be postponed indefinitely.

4. Mycosis Fungoides (Granuloma Fungoides).

This rare disease occurs usually in middle life, and is more common in men than in women. It is not hereditary. It is possibly a chronic infection, but its infectious nature has not yet been demonstrated.

In the first stage, there are macules or patches which resemble erythema, urticaria or eczema, and occur specially on the trunk, face and scalp. They soon become elevated, and, moreover, they appear and disappear spontaneously and quickly.

In the second stage, the true skin becomes more deeply involved, and the patches become more prominent. Rapid disappearance of lesions is still a feature.

The third stage is characterised by the development of sessile tumours varying in size, but occasionally larger than an orange. Some of these tumours disappear, but new patches and new tumours continue to develop. Some of the tumours now ulcerate and give rise to foul sores. At the same time any subjective symptoms, such as pain, itching, etc., which may previously have been present, disappear, but the general health begins to deteriorate.

Prognosis.—The patient usually dies after a number of years from exhaustion, diarrhœa, septicæmia, or some other complication. In rare instances recovery has taken place.

Treatment.—Arsenic, salicin, continued purgation, and exposure to the X rays, appear to be of service in occasional cases.

5. Molluscum Contagiosum.

This is chiefly a disease of children, although it may, for instance, be inoculated by a suckling on its mother's breast. In children it is most common on the face and neck. It is not uncommon about the genitals of prostitutes and of those who consort with them. It takes the form of round painless tumours which slowly grow till they reach the size of a pea. They are scattered about the skin in a sparing manner, and are usually sessile. They die out spontaneously and are succeeded by others. Each tumour has a thick wall, and on the summit there is a depression through which it is possible to express the whitish semifluid contents resulting from degeneration of the epithelial cells of the growth. The degenerative changes in some of the cells account for the so-called 'molluscum bodies,' which were at one time supposed to be a manifestation of infection by protozoa. The tumours take origin in the Malpighian layer of the epidermis.

Treatment.—It may be sufficient to squeeze out the contents of each tumour after making a small cut in its substance; but it is better, after the incision and evacuation, to touch the part with nitric acid, with pure carbolic acid, or with the galvano-cautery.

6. Molluscum Fibrosum (Fibroma Molluscum).

This disease is characterised by the development of small fibrous tumours which take origin in the connective tissue of the skin or subcutaneous tissue. It is believed that many of these growths are congenital; and in any case they are usually first observed in childhood, though they may develop further at a later period. The tumours vary greatly in shape

and size, and may be either sessile or pedunculated. A large proportion of people have one or two growths of this kind on their bodies. Small ones are usually soft, and may feel like empty bags of skin, but large ones are generally firmer. In rare cases, these tumours are present in enormous numbers and cause great disfigurement. The disease does not give rise to subjective symptoms.

Prognosis.—Fibroma molluscum is not malignant and tends to become stationary after a time.

Treatment.—Any treatment required must be surgical.

7. Xanthoma (Xanthelasma. Vitiligoidea).

In this disease well-defined yellow patches appear in the skin. The principal change is in the deeper layers of the true skin, where an overgrowth of connective tissue takes place. Between the fibres of this tissue, epithelioid cells of various sizes are present, and in the cells are yellow pigment, fat, and crystals of cholesterin and tyrosin. There is much fat also in the tissue outside the cells. The disease is met with in different forms. The first two described here are more common in women than in men ; the third or diabetic variety is much more frequent in males.

Xanthoma Planum (*Vitiligoidea plana*) is an affection of the second half of life, and is most common in and near the eyelids, often forming a half-circle around the canthus. It is generally bilateral. It assumes the form of smooth, flat, yellowish, well-defined patches which are quite soft and elastic, and look like pieces of chamois leather let into the skin. After growing to some size, they may remain stationary for an indefinite number of years. There are no subjective symptoms.

It is very seldom that the patches disappear, but in one case which came under my observation many years ago, where a large patch surrounded the left inner canthus and involved both upper and lower lids, the lesion had almost completely disappeared within three years.

Xanthoma Tuberosum (*X. multiplex, Vitiligoidea tuberosa*) is much less common, but is more important than *X. planum.*

It is met with chiefly in individuals who have been suffering for a prolonged period from jaundice. It is generally preceded by flat xanthoma of the eyelids (already described), which may thus seem to spread to other parts of the face and the body. The eruption consists of numerous little papules which are best seen on the limbs. The disease progresses very slowly, and may involve the mucous and serous membranes. A few cases have been met with in children, but never in association with jaundice.

Xanthoma Diabeticorum (*X. glycosuricum*, *Lichen diabeticus*) is a rare condition which occurs almost solely in individuals who are actually glycosuric. The other symptoms of diabetes mellitus are not always present. Alcoholism and obesity are sometimes associated facts.

The eruption is most common on the buttocks, elbows and knees. It develops rather quickly, and after remaining in a stationary condition for months or years, passes away completely. The nodules are at first bright red, but afterwards become yellow. They are sometimes tender at first, and their appearance may be preceded by neuralgia. Itching and tingling are other subjective phenomena which are present in many cases.

Treatment of Xanthoma.—*Xanthoma planum* and *X. multiplex* do not require treatment except on account of disfigurement of the face. Patches in the eyelids are best removed by excision, while those in other parts may be treated by the X rays. In *X. diabeticorum*, cure of the glycosuria is likely to be followed by disappearance of the eruption. Local soothing agents may be used to relieve itching.

8. Cheloid (Keloid, Cancroid, Alibert's Keloid).[1]

A distinction was formerly made between true (spontaneous or idiopathic) cheloid and false (spurious, cicatricial or scar) cheloid, but there appears to be no essential difference between the two. Moreover, a supposed idiopathic cheloid

[1] Addison's keloid is morphœa or circumscribed sclerodermia (p. 930).

may well have originated in a minute scar (*e.g.*, from acne) which had been overlooked.

Cheloid is due to multiplication of connective tissue cells with fibrous tissue development in the deeper part of the true skin. The epidermis remains intact throughout, and the papillæ are, for a time at least, unaffected. The disease may occur at any age, and may attack any part, but is most common over the sternum. The affected area is elevated and has an abrupt margin, from which branches suggestive of a crab's claws are thrown out. The overlying skin is tense and shiny. It may have a pink colour and may show dilated vessels. The growth is firm, often tender, and often itchy or painful. It may be multiple.

Prognosis.—The growth generally recurs after excision, but it remains a strictly local affection.

Treatment.—The seat of disease should be protected from irritation by a plaster. Itching or pain may require to be relieved by local or internal medication. If it is desired to remove the cheloid the X rays may be employed, or thiosinamin (20 minims of a warm 10 per cent. solution in equal parts of glycerin and water) may be injected locally from time to time.

iii. Diseases due to Parasitic Fungi.[1]

1. Ringworm.

Until a few years ago, all the ringworms were supposed to be due to one fungus, *Trichophyton tonsurans*, and ringworm in general was called *Tinea*[2] *trichophytina*. It is now recognised that the ringworms are due to various species of microsporon and to various species of trichophyton. Some of these parasites are peculiar to man, while others are derived from the lower animals. The former readily spread from person to person, especially among children, either by direct contact or indirectly.

Of the *microspora*, *Trichophyton microsporon*, or *Micro-*

[1] Diseases due to animal parasites are considered in Section XII.

[2] Tinea (Lat., *moth* or *worm*) is a generic name for skin diseases due to parasitic fungi.

sporon audouini is important. As seen in an affected hair, its spores are small, and quite irregularly arranged ('small-spored fungus').

Of the *trichophyta*, *Trichophyton megalosporon* ('large-spored fungus') deserves mention, with its varieties known as the *endothrix* and *ectothrix* according as the fungus is inside or outside the hair. Its spores are arranged in chains.

The ringworm fungi belong to the moulds. All the three kinds mentioned may attack the scalp and the smooth skin. The ectothrix attacks the beard. The ectothrix, and occasionally the endothrix, attack the nails.

Ringworm of the Scalp (*Tinea tonsurans*) is in Scotland and London almost always due to the microsporon, though sometimes to the endothrix trichophyton. This arrangement, however, does not hold good in all countries. Thus it is said that in the elementary schools of Paris, the microsporon and the endothrix trichophyton are almost equally common, while in Italy the trichophyton is almost always the organism present. The disease is generally acquired after the commencement of school age, and it dies out spontaneously about puberty.

Symptoms.—Ringworm of the scalp is first recognised by the development on the head of a small bare-looking spot; and closer inspection shows that the hairs in this spot are broken, bent and inelastic. The skin is scaly and occasionally reddened. The development of numerous coalescing patches may lead to involvement of most of the scalp. An attempt to pull out a bunch of the stumps causes many of them to break off in their follicles. A grey sheath of spores may extend up the outside of the hairs for a perceptible distance above the follicles. The fungus may be detected by examining the hairs microscopically, after soaking them for half an hour in liquor potassæ.

The microsporon occasionally attacks the smooth skin, either alone or along with the scalp. Its inflammatory effects on the skin are usually trifling compared with those of the megalosporon. It does not attack the beard or nails.

Diagnosis.—In *alopecia areata* (p. 887) the affected patch is smooth, and there is no scaliness or breaking of hairs.

Treatment.—This is very troublesome, because of the difficulty in reaching the fungus with a parasiticide. If there is only one patch, the hair should be cut short around it. If there are several patches, all the hair, except possibly a fringe at the margin, should be cut short or shaved off. The head should be thoroughly cleansed at the outset, and from time to time afterwards, with the aid of soft soap. Glycerin of carbolic acid or some other parasiticide should be applied daily to prevent auto-inoculation, and a cap should be worn night and day to prevent contamination of the pillow, hat, etc.

Parasiticides are practically useless for destroying the fungus in the hair-follicles; the only resource is to cause such inflammation of the hair-follicles as will loosen the attachment of the diseased hairs and allow them to fall or be pulled out. Epilation would be effectual if it were practicable, but unfortunately the hairs usually break when an attempt is made to pull them out. A parasiticide, therefore, which will cause deep-seated folliculitis is the remedy indicated, and the physician must aim at producing sufficient but not too much inflammation. An ointment of oleate of copper (ʒi.-ʒiv. in ʒi.) is strongly recommended by Crocker. It either causes the diseased hairs to fall out, or facilitates epilation. It occasionally produces a slight *kerion* (see below), which is a desirable effect. Another method consists in repeated applications of croton oil over a small area, with continuous poulticing. This gives rise to an artificial kerion, but should cause no scarring. Colcott Fox recommends as an alternative the inunction of a parasiticide ointment containing 1 drachm of croton oil per ounce. The X rays may be used for the purpose of epilation. Cure is indicated by the growth of new hairs and the persistent absence of diseased hairs.

Kerion[1] (*Tinea kèrion, Kerion Celsi*) is a peculiar variety of ringworm, which, it would appear, is specially, though not exclusively, associated with trichophyta of animal origin, and particularly with *T. megalosporon ectothrix*. The fungus sets up a severe folliculitis, and the affected patch is swollen and boggy. The follicles are dilated and discharge a sticky,

[1] Greek, *honeycomb*.

transparent or sero-purulent fluid, so that the appearance of a honeycomb is suggested.

Treatment.—The swelling must not be opened. The hairs fall out, and with them the cause of the disease, so that spontaneous healing of that particular patch is the result. If there is much inflammation, antiseptic fomentations may be applied, and an effort should be made to prevent neighbouring parts from being infected.

Ringworm of the Body (*T. circinata*) may be due to any of the ringworm fungi. These grow in the skin and set up inflammatory reaction. Red circular areas are produced which may either be scaly or present vesicles or pustules. These areas grow in size, and heal at their centre, so that rings are produced; whence the name of the disease. The megalosporon causes much more inflammation than the microsporon, and may produce not only crusting but even vesication and pustulation. If pustules occur, the fungus is pretty sure to be an ectothrix of animal origin.

Kerion (*agminate pustular folliculitis*) may develop on the backs of the hands and forearms, and occasionally elsewhere. It occurs chiefly in those who have to do with animals. Pus exudes from the follicles, and the diseased hairs become detached, so that cure results.

Tinea inguinalis, cruris, seu axillaris, is ringworm as observed on the genitals, the upper parts of the thighs and the axillæ. The affected part is red and itchy, and has a well-defined margin. This condition is due to the megalosporon, and constitutes one of the diseases formerly known as *Eczema marginatum*.

Tropical ringworm often begins in the inguinal region, but is apt to spread widely. It is seen chiefly in adults. The secondary inflammation of the skin which may result from sweating and scratching gives rise to much annoyance.

Treatment.—Ringworm of the body is easily cured by causing desquamation of the superficial layers of the skin in which the fungus grows. Repeated applications of tincture of iodine or some parasiticide ointment are sufficient.

Ringworm of the Beard (*T. barbæ*, *T. sycosis*[1]) may present itself as an itchy, red, scaly ring, which may be associated with papules, vesicles and even a few pustules. In the more severe or kerion form (formerly called *Sycosis menti*) there is a deep-seated folliculitis which gives rise to a painful, nodular swelling. There is almost always a history of a 'foul shave' in a barber's shop, but the fungus (viz., the ectothrix) appears to come originally from the horse, cow, or some other lower animal. The hairs do not break off so readily as in ringworm of the scalp.

Treatment.—The hairs on the affected parts must be thoroughly removed. Thereafter a mild case may be treated in the same way as *T. circinata*, while a severe case will be treated in the same way as kerion of the scalp (*e.g.*, with oleate of copper ointment, ʒss. to ʒi.).

Ringworm of the Nails (*T. unguium*, *Onychomycosis*[2]) is rare and is said to be always due to trichophyta derived from the lower animals. The affected nails become thick, opaque and brittle. The disease is extremely obstinate.

2. Tinea Favosa (Favus. Porrigo Favosa. Honeycomb Ringworm).

Favus attacks chiefly the scalp, but may also affect the smooth or glabrous skin and the nails. The fungus is *Achorion Schönleinii*. The disease seldom commences after twenty, but may persist for an indefinite number of years. It is transmitted from person to person, or from some lower animal to mankind—for instance, from mice to cats, and from cats to man. Its frequency varies much in different countries.

Having gained access to the *scalp*, the fungus soon attacks the hair-follicle and hair. A little sulphur-yellow disc or *scutulum* forms in the substance of the epidermis, over the affected hair-follicle. The crust is concave in its centre

[1] It is important to bear in mind that *T. sycosis* (*T. barbæ*, *Sycosis menti*, *Hyphogenic sycosis*) is an entirely different disease from *Sycosis* (*Coccogenic sycosis*). The latter is a suppurative folliculitis of the beard region (p. 924).

[2] The term *Onychomycosis* is also applicable to favus of the nails.

above, convex below, perforated by one or two hairs, and surrounded by an inflammatory areola. The crust is perhaps a quarter of an inch in diameter. At first it is quite soft, but it becomes firm with age. It lies in a cup-like depression, which is red and moist, and has a hair in its centre. If the cup is removed, another develops in its place, unless the hair is extracted in its entirety and a parasiticide is applied. The discs may remain discrete, or become confluent. The hairs are brittle, but not so much so as in ringworm. In the absence of treatment, the disease tends to cause destruction of the follicles and permanent baldness. There is usually some itching, and the crusts give off an odour suggestive of mice, or of the urine of cats.

The microscope reveals the presence of the fungus in the form of spores and mycelium. The parasite is found in the lower part of the hairs, in the epidermis, and on the free surface of the skin.

When favus attacks the *glabrous parts*, it may give rise to an appearance indistinguishable from that of *Tinea circinata*, although sometimes yellowish streaks may excite suspicion, or an occasional favus cup may make the nature of the disease evident.

Favus of the *nails*, which is extremely rare, may exactly resemble ringworm. Even on the scalp, the characteristic crusts are occasionally absent.

Treatment.—This is difficult. Removal of the crusts and thorough epilation, in combination with parasiticide applications, constitute the effectual remedy. Fortunately the diseased hairs are not very brittle, so that they can be extracted entire.

3. Tinea Versicolor (Pityriasis Versicolor).

This disease is due to the presence in the cuticle of *Microsporon furfur*. It is most common on the trunk, and occurs in the form of patches of a fawn colour. These patches may be surrounded by detached spots of the same colour, indicating the spread of the disease. In some parts a very scanty fine desquamation will be recognisable, and

this becomes more apparent on scratching. If some of the scales thus scratched off be put on a slide, and be then moistened with a drop of liquor potassæ and placed under the microscope, it is easy to recognise the fungus with its branching tubes, and its spores arranged like bunches of grapes.

There are few or no subjective symptoms. The disease occurs in adults, and chiefly in phthisical persons and others who constantly wear very warm clothing and sweat freely.

Treatment.—The clothing should be disinfected. The affected parts should be scrubbed each evening with soap and hot water, and thereafter a lotion containing perchloride of mercury (gr. ii. in ℥i.) with a little glycerin should be well rubbed in.

4. Tinea Imbricata (Tinea Desquamans. Tokelau Ringworm).

This disease is endemic in certain tropical countries, and may attack any part of the body except the head. Its most characteristic feature is the occurrence of desquamation in concentric and ever-growing rings which start, one after another, from each seat of inoculation where the fungus is growing in the skin. The scales of each circle are attached at their peripheral margin, and are free and get broken off at their inner margin. There is little or no inflammation or itching. The fungus is present in the scales in great abundance.

Treatment.—The application of iodine liniment or of sulphur ointment, together with disinfection of the clothing, removes the disease.

5. Erythrasma.[1]

Erythrasma is due to an extremely minute fungus, *Microsporon minutissimum*. The parasite gives rise to rounded,

[1] Erythrasma is sometimes (like *Tinea inguinalis*) included under *Eczema marginatum*.

brownish patches, which are most common in men on the scrotum and neighbouring parts of the thigh. It sometimes attacks the corresponding regions in women, and the axilla in either sex. It is distinguishable from *tinea cruris* by the absence of inflammatory phenomena. It causes almost no trouble, and may be cured by parasiticide applications.

SECTION XI

INTOXICATIONS AND SUNSTROKE

1. ALCOHOLISM

Acute Alcoholic Intoxication or drunkenness scarcely comes within the province of the physician, but it must be borne in mind as an occasional cause of fatal coma with or without convulsions. The diagnosis from apoplexy and uræmia is of great practical importance (see p. 765).

Delirium Tremens (Acute Alcoholism).

Delirium tremens may be primary or secondary. The *primary* form occurs in habitual drunkards. The *secondary* or associated form (*alcoholic delirium*) is called forth by acute disease (*e.g.*, pneumonia or erysipelas), or by the shock of an injury, in an individual who takes large quantities of alcohol, but who may, nevertheless, to all appearance, be a temperate man. Delirium tremens occurs chiefly in middle life, and is far more common in men than in women. The attack generally follows a severe drinking bout. As the bout may have ceased some days before the delirium sets in, there is a popular notion that the delirium is due to the discarding of the alcohol. Another explanation is that the patient stops drinking because with the commencement of the attack a distaste for alcohol sets in.

Morbid Anatomy.—There is congestion of the cerebral cortex. Other changes which may be discovered are attributable to the chronic alcoholism, or to associated disease, or to the mode of death.

Symptoms.—The symptoms take two or three days to reach their height. They include anorexia, restlessness and insomnia; horrible dreams, if sleep should come; and terrifying visual hallucinations in the waking period. The patient imagines he sees living creatures like rats and demons. Auditory hallucinations also may occur, so that the patient thinks that those around are insulting him. Delusions and suicidal impulses are sometimes met with. The patient is very talkative, but is incoherent. Well-marked tremor attends voluntary movements, especially in the face, tongue and upper limbs. The pulse is soft and accelerated, and pyrexia is present. The tongue is thickly furred, and there is abundant perspiration.

After several days, the patient in the most favourable cases has a prolonged sleep and wakes up practically well, though the tremor may continue for some time. In other cases, the subsidence of the attack is gradual, the early periods of sleep short, and prostration considerable and dangerous. Insomnia remains absolute in some cases, till death takes place from exhaustion.

Diagnosis.—The condition is a well-marked variety of melancholia of the excited type. The peculiar character of the delirium, the tremor, and the habits of the patient, make the diagnosis easy. The most important point is to examine the body, and especially the lungs, for any local disease or injury.

Prognosis.—Most cases recover within a week. Death may be due to exhaustion or to associated disease—*e.g.*, pneumonia or tuberculosis. Recurrence is common, and a second or third attack is more dangerous than the first. It is probable that many suicides and murders are due to commencing delirium tremens.

Treatment.—The patient must be prevented from injuring himself, and must be fed at least once in three hours, with liquid nourishment, such as milk, egg-flip and soup. The bowels should be freely opened at the outset. If the pulse is feeble, ammonia and strychnine should be employed as stimulants rather than alcohol. Hypnotics must be employed with great caution. The patient is very insusceptible to their

influence, but if the dose is frequently repeated, a fatal cumulative effect may at last be produced. If the patient has had no sleep by the third or fourth day, potassium bromide (gr. xxx.), chloral (gr. xv.), and tincture of capsicum (♏ xxx.) may be given every eight hours. Or paraldehyde in doses of 2 drachms or more may be given in combination with bromide. Opiates should be avoided. If the patient will not swallow, the remedies recommended for administration by the mouth may be given by the rectum ; or hyoscine hydrobromide ($\frac{1}{150}$ grain) may be injected hypodermically.

Acute Alcoholic Insanity is an occasional result of alcoholism. Thus in young subjects who have a hereditary instability of brain organisation, an attack of acute mania may be induced by a few glasses of spirits (*mania a potu*). Acute melancholia may also be due to alcohol.

Chronic Alcoholism.

Chronic alcoholism manifests itself in many ways. Thus, in the case of the *digestive system*, there is chronic gastric catarrh with anorexia, furring and tremor of the tongue, morning sickness and irregularity of the bowels. Cirrhosis of the liver may occur, and as a consequence hæmatemesis, melæna, piles, distension of the small bloodvessels on the face, acne rosacea, yellowness of the conjunctivæ, and ascites. Or the liver may become fatty and enlarged.

Disease of the *kidneys* is generally recognised as being one result of chronic alcoholic excess. The *heart* and *blood-vessels* also suffer, the latter becoming sclerosed, and the former being dilated and showing fatty degeneration or fatty infiltration.

Of all the systems, however, the *nervous system* is that which is most seriously involved. In ordinary acute alcoholic intoxication, its functions are the first to be obviously deranged, as shown in the early stages by inco-ordination, loquacity and excitement, and at a later stage by stupor. In chronic alcoholism, muscular tremor on voluntary effort is a common symptom, involving especially the hands, lips and tongue. Both mind and morals deteriorate. The memory fails ; the patient is restless, irritable and unre-

liable ; he sleeps badly, and is troubled with unpleasant dreams. Hallucinations of hearing, delusions of suspicion, assaults or actual murders and suicide also occur. Chronic dementia may supervene with distinct diminution of muscular power.

Important structural changes may occur, including multiple neuritis, retrobulbar neuritis (toxic amblyopia), myelitis and meningitis. Chronic alcoholism is an occasional cause of epilepsy, and of various kinds of insanity.

Prognosis.—As a rule the patient continues to drink until he dies, after a few years, from disease of the liver, nerves, lungs or other viscera. Some cases are rescued before permanent damage is done, and are restored to health and fitness for duty. In others, again, the morbid changes are arrested, and even in part recovered from, but the patient does not completely regain his normal condition.

Treatment.—This must begin with complete abstinence from alcohol, a condition which is very difficult of attainment unless in an institution. The patient should have a tonic containing nux vomica (℥ v.), aromatic spirit of ammonia (℥ss.), and tincture of capsicum (℥ x.), a formula which may do something to allay the craving. Bromide, Indian hemp, hyoscyamus or paraldehyde may be given at bedtime with a glass of hot milk for insomnia. It will help much if the sufferer's interest can be thoroughly engaged by some hobby of a healthy character.

2. MORPHINISM

(Morphine Habit. Morphinomania).

The habitual use of opium or morphine is generally commenced for the relief of pain. As much as a pint of laudanum has been known to be taken daily. De Quincey, who gave up opium 'after an eighteen years' use, and an eight years' abuse of its powers,' took at one period 320 grains a day.[1] Considerable doses may be taken by some

[1] 'Confessions of an English Opium-Eater.' Lauder Brunton estimates De Quincey's daily allowance of 9 ounces of laudanum as equivalent to 333 grains of solid opium. Coleridge had a desperate struggle before he got rid of the opium habit.

people for a long time without obvious disturbance of the health. But very often the quantity requires to be increased, and as the effect of each dose wears off, the victim is driven by a sense of physical and mental prostration to resort to another.

Symptoms.—The opium-eater is pale or sallow, and becomes prematurely grey and old-looking. His eyes are sunken, his expression dreamy, and his body emaciated. The appetite and bowels are irregular. Shortly after a dose there may be increased intellectual activity, but the memory tends to suffer, and the moral nature may undergo great deterioration, as is shown, for instance, by the devices adopted to conceal and to perpetuate the habit, by the untruthfulness, and by the neglect of duty. Sudden withdrawal of the drug often causes great suffering; the symptoms include diarrhœa, restlessness, and insomnia. In suspected cases, if the practice is denied, the arms and other parts should be examined for marks of the hypodermic syringe.

Treatment.—Treatment should be carried out in an institution, and the drug should be withdrawn gradually. After the daily dose taken by the syringe has been greatly reduced, the further gradual reduction of the hypodermic dose may be accompanied by rectal administration. After the hypodermic dose has been reduced to zero, the rectal dose may be gradually reduced to zero also. Jennings, who employs rectal administration, calls attention to three results of the withdrawal of morphine and to the corresponding remedies: (1) restlessness, or even pain, which is due to lack of the artificial stimulus hitherto administered to the brain cells; (2) feebleness of the circulation; and (3) excessive secretion of acid in the stomach. The respective remedies are: (1) hot-air baths; (2) cardiac tonics; and (3) bicarbonate of sodium. Occasionally, however, a patient is able to dispense with the drug, and apparently without difficulty, by a complete change in his surroundings; for instance by living in the Highlands, with good society, a bracing atmosphere, and plenty of open-air amusements.

3. LEAD POISONING

(Plumbism. Saturnism).

Lead may enter the system by the alimentary tract, the respiratory tract, or the skin. It is often taken by women to produce abortion. Poisoning sometimes occurs through the drinking of contaminated water, and very often as a result of an occupation which involves the handling or inhalation of lead salts. Thus lead smelters, white lead and red lead workers, painters, compositors, potters and others are liable to suffer. Individuals vary greatly in their susceptibility. The female sex, alcoholism and gout predispose to plumbism.

Saturnine Cachexia is characterised by anæmia and an earthy tint of skin. According to Gowers, the red corpuscles are reduced in proportion to the hæmoglobin, so that the anæmia resembles that of pernicious anæmia and not that of chlorosis. It is perhaps attributable to accumulation of lead in the bone-marrow. There is a bad taste in the mouth, the breath is offensive, and a dark-blue line is seen at the margins of the gums. This is due to sulphide of lead which is deposited in the gum close to its deep surface. The sulphuretted hydrogen which combines with the lead is derived from the decomposition of albuminous food which collects where there is a slight separation between gum and tooth. There is no lead line where the teeth are absent. There may be pains in the joints, muscles or nerves ; tenderness of the muscles ; and occasionally tingling in the limbs, and patches of impaired sensation.

Lead Colic (*saturnine* or *painter's colic*) is characterised by severe abdominal pain which is generally associated with retraction of the abdomen and constipation, and a slow pulse of high tension. The pain is paroxysmal and affects chiefly the umbilical region. It is supposed to be due to cramp of the colon.

Lead Palsy is common, and is generally accompanied by little or no sensory disturbance. The usual form is symmetrical wrist-drop of *acute* or *subacute* onset. The muscles involved are the common extensor of the fingers, the extensor indicis, the extensors of the phalanges of the

thumbs, and the extensors of the wrist. The supinator longus and usually also the extensor of the metacarpal bone of the thumb escape, though supplied, like the others, from the musculospiral nerve. In a few cases, this form of paralysis attacks the muscles of the upper arm, of the hand, or of the leg. When the legs suffer, the muscles chiefly involved are those which are homologous with the affected forearm muscles, namely, the long extensor of the toes and the peronei, which are supplied by the peroneal or external popliteal nerve. The tibialis anticus, however, though also supplied by that nerve, usually escapes, like the supinator longus in the upper limb. This palsy is accompanied by wasting, and by the degenerative reaction. It is probably due to neuritis of the motor nerve fibres, and, with appropriate treatment, may recover perfectly.

A second form is *chronic*, and has little tendency to recover. It occurs along with the other, or by itself. It resembles progressive muscular atrophy, and begins especially in the intrinsic muscles of the hands, though it may spread widely. In this form weakness and wasting keep pace with one another.

Saturnine Encephalopathy assumes varied forms. There may be convulsions, delirium and coma of acute onset, often accompanied by optic neuritis. The patient may die from the cerebral disturbance, or may be rendered permanently blind by the neuritis. Female lead-workers may be attacked by symptoms which appear to be hysterical and of quite trifling severity, and may yet be dead in a couple of days. Melancholia with delusions, and other mental symptoms may occur.

Kidney Disease is a common result of chronic lead poisoning. There is at first a parenchymatous inflammation which tends later on to become interstitial ; so that in chronic cases, the kidneys are pale though contracted. Gout and lead poisoning predispose to one another. Lead interferes with the excretion of uric acid.

Arterio-sclerosis and cardiac hypertrophy are natural consequences of chronic kidney disease, with or without gout.

Menorrhagia and Miscarriages are common in female lead-workers. Oliver states that it is almost impossible for a pregnant female lead-worker, even though she has no symptoms of plumbism, to continue her occupation without aborting.

Diagnosis.—In acute cases the sudden onset of colic, the blue line on the gums, and the occupation or the history, make the diagnosis easy. Where there is no history of contact with lead, and no blue line is present on the gums, and where the principal complaint is of a gradual deterioration of the general health, the true nature of the illness may easily be overlooked. In such a case, there may be a history of colic ; constipation and anæmia will be present ; the patient may be conscious of a metallic taste in the mouth ; and females may have had menorrhagia or miscarriages. Chemical examination of the urine may reveal the presence of lead, although it has been found that such a discovery is not an absolute proof of chronic lead poisoning. The test may be carried out by placing a strip of magnesium in the suspected urine, and adding ammonium oxalate in the proportion of 1 gramme to 150 c.c. of the urine. If lead is present, it is deposited on the magnesium, and will probably be visible within half an hour. The slip is then washed in distilled water and dried, and the deposit is submitted to confirmatory tests. Thus when the slip is warmed with a crystal of iodine upon it, the development of a yellow colour shows that lead is present. Or the deposit may be dissolved in nitric acid, and the solution is then tested for lead in the usual manner.

In *alcoholic neuritis*, sensory as well as motor nerves are affected, and the lower limbs suffer more than the upper.

Pressure-paralysis involving the muscles supplied by the musculospiral nerve is unilateral.

Treatment.—Much of the treatment is symptomatic. The patient must of course withdraw from his work. Colic may require morphine hypodermically and fomentations locally. Constipation should be treated by Epsom salts with dilute sulphuric acid. Iron may be given for the anæmia.

When all active symptoms are past, iodide of potassium

should be given in small doses (gr. iii.-v. t.i.d.) to convert the lead deposited in the tissues into a soluble salt, so that it may be taken up again into the circulation and gradually eliminated.

In the convulsions of saturnine encephalopathy, nitrite of amyl should be administered by inhalation.

In acute paralysis, electricity should be applied to the muscles to assist in maintaining their nutrition.

At a later stage, massage and the internal or hypodermic use of strychnine will be indicated.

4. CHRONIC MERCURIAL POISONING

(Hydrargyria. Mercurialism).

This condition is met with in those who work in quicksilver mines; in makers of thermometers, barometers, mirrors, etc.; and in others who breathe mercurial vapour.

Symptoms.—These usually begin in connection with the digestive tract, and include slight stomatitis, gastric disturbance and diarrhœa, with anæmia and occasionally salivation. In a few cases there is a blue line on the gums which closely resembles that produced by lead.

The most characteristic symptom, however, is that known as 'mercurial tremor,' 'metallic tremor,' or 'the trembles.' At first it is observed only on movement, or when the patient is excited; but it may afterwards become continuous, though it is always increased by emotion. It begins in the face and tongue, and afterwards extends to the upper, and then occasionally to the lower limbs. It interferes with speaking and walking, and may render the patient unable to feed himself. Sensory and mental symptoms are sometimes present.

Diagnosis.—In *insular sclerosis*, there is not likely to be a history of exposure to the vapour of mercury; and there is frequently partial palsy of one or more limbs, with sensory disturbance and nystagmus.

In *paralysis agitans*, the tremor is fine and rhythmical, and continues when no voluntary movement is being executed.

The gait, the expression of face, and the muscular rigidity are other important points in shaking palsy.

Prognosis.—Recovery is the rule, if the patient keeps away from mercury.

Treatment.—Withdrawal from mercury is the essential matter. Iodide of potassium may after a time be given in small doses (as in chronic lead poisoning) to promote elimination.

5. CHRONIC ARSENICAL POISONING.

Certain bright-green and brown wall-papers contain arsenic, and persons living in rooms papered with these may be poisoned. Paperhangers, milliners, makers of artificial flowers and others are also liable. Many recent cases of arsenical poisoning in England were traced to contaminated beer.

Symptoms.—Among the results of chronic poisoning are anorexia, irregularity of the bowels, puffiness of the eyelids, conjunctivitis, pigmentation of the skin, herpes zoster and multiple neuritis. Gowers points out that the effects of arsenical multiple neuritis may assume three aspects: (1) Palsy of the muscles of the limbs (as in lead poisoning, but with more marked sensory symptoms); (2) ataxy with defective sensibility, especially in the muscles (pseudo-tabes, with inco-ordination as in tabes); and (3) sensory symptoms, namely, pain, anæsthesia, etc. (as in alcoholic neuritis).

Arsenical neuritis usually affects the legs sooner than the arms. Arsenical pigmentation is met with in many epileptic patients who have taken arsenic during a period of years to prevent the acne which would otherwise be produced by the bromide. It is often most marked on the trunk, but may be seen on the neck and limbs.

6. FOOD POISONING.

Articles of food may be poisoned in various ways. Flesh may be poisoned by drugs or food eaten by the animal. Honey may be poisonous through being derived from poisonous plants. Poison may be added to food as an

adulterant, as when tinned peas, etc., are coloured by the addition of copper salts. Certain fish and shell-fish, *e.g.*, salmon and mussels, may develop poisons in the fresh state ; whilst various kinds of animal food may develop poisons through fermentation or putrefaction. Foods may contain pathogenic microbes, or they may contain poison (*e.g.*, lead) derived from the vessels in which they are kept. Beer may contain arsenic derived from impure sulphuric acid employed in preparing the brewing glucose. Water may be poisoned by lead, micro-organisms from sewage matter, etc.

Poisoning by Animal Products.

Meat.—Poisoning by meat which has been kept too long may be due to a microbe, or to a chemical poison which the microbe produces. If the symptoms set in very shortly after the food is taken, they are probably due to the chemical poison being already present in the food ; whereas, if there is a distinct incubation period, it is probable that the organism is present in the food, and evolves its poison after the food is swallowed. Of the various kinds of meat, swine's flesh is most often poisonous.

Symptoms.—These point to severe gastro-enteritis, and include abdominal pain, vomiting, diarrhœa with offensive stools, great prostration, and commonly fever.

Treatment.—Luff recommends that the following draught be given every three or four hours : Liq. hydrarg. perchlor. ℥xx. ; pot. iod., gr. v. ; chloral hydr., gr. v. ; ac. carbol., gr. i. ; sp. ammon. aromta., ℥xx. ; aq. chlorof., ad ʒi. S. et M.

Sausages.—*Botulism* or *allantiasis* is due to poisoning by decomposition products in sausages. The microbe is the *Bacillus botulinus*, and the symptoms include dryness of the mouth and pharynx, a barking cough, hoarseness of the voice, dysphagia, a rapid pulse, dilatation of the pupils, vomiting and diarrhœa, or sometimes colic and constipation. The symptoms commonly set in after twelve or more hours from the eating of the poisonous sausage. In the **treatment** of such cases, one of the first things to be done is to clear out the stomach and bowels, unless spontaneous vomiting and diarrhœa are already present. Warmth should be

applied to the surface, and morphine may be needed to allay pain and shock.

Fish and Shell-Fish.—Shell-fish, and especially mussels, may cause urticaria or gastro-intestinal disturbance. Mussels contaminated by sewage may contain a ptomaine produced by bacteria, and ingestion of such mussels occasionally causes death. In the case of fish, the poison may be due to bacteria, or may be a normal secretion of the animal's body.

Milk and Milk Products.—Milk which has undergone lactic acid fermentation may cause digestive disturbance in children. Milk may contain the germs of enteric fever, tuberculosis and various other infections. Cream, ice-cream, butter and cheese occasionally produce ptomaine poisoning.

Poisoning by Vegetable Products.

Ergotism (*Morbus cerealis*) is due to the use of meal or bread made from rye which contains ergot, the sclerotium of the fungus called *Claviceps purpurea*. In fatal cases, the arterioles show hyaline transformation of the intima, and their lumen is narrowed, blocked by thrombosis, or completely occluded. Gangrene of the extremities is a result of these changes. The posterior columns of the cord are sclerosed.

Symptoms.—There are signs of irritant poisoning—vomiting, diarrhœa, headache, and abdominal pains. Other symptoms vary in different cases. In the *gangrenous* variety the fingers and toes, and sometimes the nose and ears, become the seat of gangrene ; whilst in the *spasmodic* or *convulsive* variety, there are cramps or tetanic spasms in the limbs, and sometimes general convulsions and mental symptoms. Ergotism may be *acute* or *chronic*, and may last for weeks or months.

It has been supposed that the alkaloid cornutine causes the motor and sensory disturbances by acting directly upon the spinal cord ; whereas the sphacelinic acid causes arterial contraction by stimulating the vasomotor centre, and causes arterial degeneration by acting directly upon the walls of the vessels.

Treatment.—This includes the careful avoidance of ergot, and sometimes the use of emetics and purgatives ; but in the main it should be palliative and symptomatic.

Lathyrism (*Lupinosis*) is due to the use of food made from the seeds of the vetch or chickpea (*Lathyrus sativus* and *L. cicera*). It occurs in Italy, France, Spain and India. There is paralysis of the legs, with rigidity, contracture, and increase of the tendon-jerks. There may also be disturbances of sensation.

Pellagra (*Maidismus*) is due to a poison taken into the system with maize, and is endemic in Italy, Spain and elsewhere. The symptoms include erythema and burning or itching of the exposed parts of the skin (*Erythema pellagrosum*), digestive disturbances, spastic paraplegia, and after a time mental derangement. Degeneration of the posterior and lateral columns of the cord has been observed after death.

7. SUNSTROKE

(Insolation. Heatstroke).

The phenomena included under this designation may be due to the heat of the sun itself or to artificial heat. They thus occur in soldiers on the march, especially if encumbered by accoutrements ; in stokers ; and in those living in hot tents, barracks, etc., in the tropics, either by night or by day. Dampness of the atmosphere, the absence of wind, and the proximity of surrounding hills render the heat less tolerable ; whereas a robust constitution, temperate habits, and acclimatisation increase the powers of resistance.

Morbid Anatomy.—Where death takes place suddenly, there may be no important changes. In cases of *thermic fever*, the lungs are often congested, and the whole venous system is engorged. The body and the internal organs may be very hot for some time after death, and rigor mortis sets in early.

Pathology.—It is natural to suppose that the symptoms are due to the heat interfering with the activity of the vasomotor and respiratory centres, but Sambon considers that *siriasis* is an infectious fever.

Symptoms.—Three types of cases are described :

(1) In the *syncopal form* (*heat exhaustion*), there is great exhaustion and prostration, from the combined influence of fatigue and exposure to a high temperature. The surface is pale and cold, and the pulse is rapid and feeble. Fatal collapse may take place, but recovery is common.

(2) The *asphyxial form* (*sunstroke* proper, *coup de soleil ;* inaccurately called ' heat apoplexy ') occurs specially when the head and spine are directly exposed to a strong sun. Asphyxia may set in rapidly, with unconsciousness, feebleness of the pulse and coldness of the surface. The attack may end in death, which may be very sudden ; or in perfect or imperfect recovery.

(3) In the *hyperpyrexial form* (*thermic fever, heat fever, siriasis*), the temperature may range from 107° to 111° F. The condition is said to set in as commonly by night as by day. The onset may be sudden or acute, and in fatal cases the end may come after minutes or hours. The symptoms include headache, vomiting, diarrhœa, a dry, hot skin, a full rapid pulse, laboured breathing, delirium, convulsions and coma. In favourable cases, the attack usually ends by crisis within one or two days. A certain amount of mental failure and persistent intolerance of heat may render the recovery incomplete.

Prognosis.—Of the three varieties of illness just described, the first is the least, and the last the most dangerous. The mortality of all kinds together is estimated at from 25 to 50 per cent., but the so-called recoveries are often incomplete.

Treatment.—In the syncopal form, the patient should be placed in a cool place with the clothing loosened. Stimulants, and even a hot bath, may be desirable. In the asphyxial form, if the patient falls down suddenly under a hot sun, the treatment is cold douching of the head and body. In thermic fever, the cold bath or ice pack must be employed. If death from asphyxia seems imminent, venesection is indicated, but if there is no urgency, this should be avoided. Chloroform may be required for convulsions. After recovery, the patient should remove to, and if possible remain in, a cool climate.

SECTION XII

DISEASES DUE TO ANIMAL PARASITES

1. PROTOZOA.

A NUMBER of diseases which attack man are attributable to parasites belonging to the protozoa. They include amœbic dysentery, malarial fever, kala-azar, trypanosomiasis, and piroplasmosis, all of which have been described among the infections (Section I.).

Among the protozoa may also be mentioned *Trichomonas vaginalis*, which is sometimes found in the mucus of the vagina ; *T.* (*Cercomonas*) *hominis*, which lives in the intestine and may be found in the stools, and *Paramœcium* (*Balantidium*) *coli*, which is occasionally present in the large intestine in cases of diarrhœa and dysentery, and has been known to invade the liver by way of the bile-ducts.

Certain parasitic protozoa which multiply by spore formation (sporozoa) give rise to the condition known, when it occurs in man, as *psorospermosis*, or *psorospermiasis ;* it resembles *coccidiosis* of the liver of the rabbit. *Coccidium oviforme* produces little nodules in the rabbit's liver. These nodules are portions of bile-ducts, whose lining epithelial cells contain the coccidia.

A similar condition, apparently due to the same parasite, is occasionally found *in man*, involving the liver, mucous membranes or skin, and in rare instances generalised throughout the viscera. The psorosperm or original unicellular organism multiplies by division. Each segment thus formed becomes enclosed by a firm wall and con-

stitutes a spore. All the spores thus produced are enclosed in one cyst or capsule. Localising phenomena may be present according as the liver, kidney or skin is involved. General infection may give rise to an acute febrile illness which ends fatally.

Different species of *Sarcocystis* occur as psorosperms in the muscles of various animals. The ovoid bodies in the muscle are called Rainey's corpuscles, and these give rise to sickle-shaped young organisms.

It is very doubtful if psorosperms have anything to do with cancer, as was at one time supposed.

2. TREMATODA (Flukes).

Distomum Hepaticum (*Fasciola hepatica*, common fluke) and Distomum Lanceolatum are liver flukes which are occasionally found in the bile-passages of man. In rare cases they cause cholangitis, enlargement of the liver, jaundice and ascites, which may terminate fatally. These flukes are common in the bile-ducts of cattle and sheep, and are the cause of 'rot' in the latter.

Distomum Sinense is the most important of the liver flukes, and is common in India, Japan, and China. It lives in the bile-ducts and gall-bladder, and causes painful enlargement of the liver, with diarrhœa, and loss of flesh and strength. In the course of years there may be distinct cachexia with dropsy.

Distomum Pulmonale (*D. ringeri*, bronchial fluke) is the cause of the *endemic hæmoptysis* (*parasitic hæmoptysis*) of Formosa, Japan and Corea. The mature parasite, which may reach two-fifths of an inch in length, lives in cavities connected with the small bronchi, and discharges its ova into these passages.

Symptoms.—There is cough with a rust-coloured or actually bloody expectoration, which may recur during many years. The ova are present in the sputum. The parasite occasionally invades the brain.

Treatment.—If cerebral symptoms arise, operative interference may be indicated; but for the disease in the lungs, no remedy seems to be available at present.

Distomum Hæmatobium (*Bilharzia hæmatobia*, blood fluke) causes the *endemic hæmaturia* of Egypt and other countries. It is remarkable among trematodes because the sexes are distinct. Part of the flat body of the male is rolled into a gutter (gynæcophorous canal), in which the cylindrical body of the female lodges during copulation. The adult worms live in the portal vein and its tributaries, and in the inferior vena cava. They are harmless to man. The ova, however, are furnished with a spine, and when they are set free into the blood, they pass by way of the blood-vessels of the bladder and rectum into the mucous membrane of those organs, and thus into their cavities. The prostate, ureter, and pelvis of the kidney may also be invaded.

Symptoms.—The irritation caused by these ova and their boring spines gives rise to symptoms of varying severity. Hæmorrhage is common, and this may be slight or may cause fatal anæmia. Inflammation of the bladder and intestine also occurs, and vesical calculus not uncommonly develops around the ova.

The larvæ are common in the Nile and other waters, and probably reach their human host chiefly through the drinking of contaminated water. But as males suffer more frequently than females, and also bathe more frequently, it is possible that the parasite may sometimes enter through the skin, anus or urethra. Having reached the alimentary canal, the larva penetrates the mucous membrane to reach the veins. It is said that half of those belonging to the lower classes in Egypt are infected.

Diagnosis.—This is made clear by the detection of the ova in the urine or stools. An ovum measures about $\frac{1}{200}$ of an inch in length, and has a spine at one end.

Treatment.—The patient should, if possible, remove to an uninfected region. Nothing is known to destroy the parasite in the living human body. Cystitis and proctitis must be relieved by hyoscyamus, enemata, etc. Fortunately the majority of cases recover.

3. CESTODA (Tapeworms).

i. Cestodes in the Intestines (Strobilus or Mature Phase).

Tænia Solium (Pork Tapeworm).

This parasite consists of a head, which is about as large as a small pin's head, a narrow neck, and a long series of segments amounting to perhaps eight hundred in number. Apart from the small head and neck, therefore, the worm has the appearance of a piece of jointed ribbon which is often 10 feet long. The head is prolonged in front to form a rostellum or proboscis which is surrounded by twenty-six chitinous hooklets in two rows. The widest part of the head is furnished with four sucking discs. The neck is very thin, and about half an inch long. Behind this, fine transverse lines begin to appear, and further back, as the worm becomes broader, the division into segments becomes more distinct. Each segment or proglottis is bisexual or hermaphrodite, and the ducts from both sets of sexual organs open at the genital papilla which is situated at one border of each proglottis. The male organs begin to appear at about the 200th segment. The testes consists of numerous vesicles scattered throughout the segment, and giving off minute ducts to join the vas deferens which, as it approaches the genital pore, passes through the penis. The two ovaries are in the posterior part of the proglottis, and the two oviducts unite and enter the globular body, into which the yolk sac, uterus and vagina also open. The spermatozoa pass from the penis into the vagina at the genital pore, and along the vagina to the globular body near the ovaries. The eggs come from the ovaries into the globular body, and are there fertilised, covered with yolk from the yolk sac, and surrounded with shell from the shell gland, after which they pass into the uterus. In the mature proglottides (from the 600th onwards), the uterus is branched like a tree, having seven or ten offshoots on each side of its main stem ; and as the branched uterus becomes distended by the accumulating ova, the other genital organs disappear.

The worm is usually attached to the duodenum or upper

jejunum, all except the head lying loose in the intestine. As the proglottides become mature, they are detached and escape from the intestine. They may wriggle about for a little, but soon die and decompose. Meanwhile the ova in the uterus develop in their interior embryos which are furnished with six hooks. Each proglottis is about half an inch long, and contains many thousands of eggs.

If either the proglottides or the eggs are swallowed by a pig (the *intermediate host*), the egg-shells are dissolved, and the embryos escape. They bore through the wall of the alimentary canal, and settle in the muscles and other organs. They now lose their hooklets and develop a cavity at their opposite end. A projection grows inwards from the wall of this cavity, and in the interior of the hollow projection a head is developed. A fibrous capsule is formed round the parasite. This stage is called the *Cysticercus cellulosæ*, 'measle' or 'bladder worm'; and pork affected in this way is described as 'measly.' The cysticercus becomes mature in three or four months, and the head is then capable of eversion, so that it projects outwards from the cyst.

If measly pork which has not been sufficiently cooked is eaten by man, the head of the cysticercus attaches itself to the mucosa of the intestine, the cyst is lost, and a tapeworm with proglottides develops. More than one worm may thus settle in the intestine.

Cysticercus in Man.—Eggs from a proglottis occasionally reach the stomach of man instead of that of the pig, and cysticerci may thus be met with in the human brain, muscles, or subcutaneous tissues. They may survive in these parts for an indefinite number of years.

Tænia Mediocanellata (T. Saginata, Unarmed or Beef Tapeworm).

This is the most common tapeworm in this country and in others where the flesh mostly eaten is that of cattle. It is longer and broader than *T. solium*, having more than a thousand segments. The head has no rostellum or hooklets, but has four large sucking discs. About eight segments are

discharged daily, and these may work their way singly through the anus apart from defæcation, so that they may be found in the patient's clothing. The arrangement of the sexual organs is similar to that in *T. solium*, but the uterus has twenty or thirty lateral offshoots on each side, and the branching is dichotomous. By the branching of the uterus, it is easy to distinguish with the unaided eye between the segments of *T. saginata* and *T. solium*, either in a spirit preparation, or in a partly dried fresh specimen.

The cysticercus stage is found in cattle, chiefly in the muscles ('measly' beef or veal). The cyst is about a third of an inch in diameter, and scarcely distinguishable from that of *T. solium*. It is not found in man.

Bothriocephalus Latus.

This is the largest tapeworm of man. It may reach 25 feet in length, and contain 3,000 or 4,000 segments. The proglottides are mostly broader than they are long. The head has neither suckers nor hooklets, but has a groove on each side. The uterus is a convoluted tube arranged like a rosette in the middle of each segment, and the genital pore is in the middle line near the anterior end of the proglottis. The ova (unlike those of the tæniæ) do not mature in the uterus, but in fresh water.

The larvæ develop in the muscles and viscera of the pike, trout and other fresh-water fish. The worm is found in Switzerland and North-East Europe.

Tænia Nana is less than an inch in length. It occurs in Italy and Sicily, especially in children. Snails are supposed to be the intermediate hosts.

Tænia Elliptica (*T. cucumerina*) is common in dogs and cats, but occasionally occurs in man. The dog-louse is the intermediate host, and the parasite may be conveyed to a child by the tongue of the dog.

Tænia Flavopunctata is common in rats, and occasional in man. Insects are the intermediate hosts.

Symptoms Caused by Tapeworms.—In many cases tapeworms give rise to no symptoms, and the first evidence of

their presence in the intestine is the escape of the proglottides in the fæces. When symptoms are present, they are usually of a vague kind. There may be uneasiness, a griping in the abdomen, nausea, a capricious appetite, irregularity of the bowels, discomfort at the anus, and restlessness at night. Various nervous symptoms have been attributed to intestinal worms, including convulsions, chorea, strabismus, melancholia and hypochondriasis, and it is quite possible that in highly neurotic individuals such results are brought about in rare instances. It is well recognised, however, that *Bothriocephalus latus* is capable of inducing a severe and even fatal anæmia which in its clinical aspects is identical with pernicious anæmia, and is possibly due to a toxin evolved by the parasite. The symptoms due to the presence of the cysticercus in man are alluded to below (Cestodes in the Tissues).

Treatment.—Careful examination of the muscles of pigs and cattle at the slaughter-house, and in particular of the muscles of the head, neck and trunk is an important preventive measure. Thorough cooking of flesh used for food renders the cysticercus harmless. All segments passed from the intestine ought to be burned.

Of remedies administered to poison the worm, the best is the liquid extract of male fern. After taking nothing into the stomach (or at most a little liquid) during the second half of one day, the patient, if an adult, should take from one to two drachms of the liquid extract early the following morning, and this should be followed, after a couple of hours, by a dose of castor oil. It is important to examine the evacuations to see that the head of the worm has been discharged. This is often not the case, and in the course of some months the segments will again appear in the stools. No doubt the head is often well protected by the *valvulæ conniventes* from contact with the poison. Other anthelmintics are pomegranate (2 ounces of the decoction every hour for three doses), kousso (4 drachms infused in boiling water, the whole being taken as soon as it is cool, without being strained), kamala (2 drachms in syrup or gruel), and tannate of pelletierine (in 2-grain doses).

ii. Cestodes in the Tissues (Scolex or Immature Phase).

It has been already mentioned (p. 961) that TÆNIA SOLIUM is occasionally found in the tissues of man as a cysticercus (*Cysticercus cellulosæ*). The parasite may reach the stomach by its own power of movement, or along with regurgitated intestinal contents in vomiting, or by being swallowed. In the case of the pig, the cysticerci do not appear to cause much trouble, and even in man they cause disturbance only by settling in important structures, or by entering the system in great numbers. In the case of man they attack the brain most commonly, the muscles much less frequently, and other organs only occasionally. When they invade the muscles and other tissues in great abundance they may give rise to rheumatic-like symptoms. When the brain is the seat of lesion, symptoms may or may not be produced. The involvement of vital structures like the medulla oblongata may cause death. Occasionally the parasite is situated in the eye, and can be recognised by the ophthalmoscope.

TÆNIA ECHINOCOCCUS gives rise to a cysticercus of man which is much more important than that of *T. solium*. The mature worm lives in the intestine of the dog, wolf and jackal, and these animals are infected by eating the flesh of sheep, pigs or cattle, in which the cysticerci are present. The tapeworm is only about a sixth of an inch long, and consists of four segments of which the last only is mature. The head has four sucking discs, a rostellum, and hooklets.

The eggs set free from the intestine of the dog are taken with the drinking-water, or occasionally with the food, into the stomachs of sheep, pigs, oxen, and in some countries mankind. The embryos which are then liberated have six hooks, and travel into the tissues. They often reach the bloodvessels, and are carried as emboli to different organs, where they develop into hydatid cysts. In man they settle most commonly in the liver, and therefore probably travel by the portal vein, but they may pass through the portal system into the hepatic veins, and thus on to the lungs and other organs.

Hydatids (*Echinococcus hominis*). — Having reached its destination, the embryo loses its hooks and is converted into a cyst, whose wall consists of two layers. The outer is a laminated chitinous membrane or cuticle (ectocyst); the inner (endocyst) is granular and parenchymatous, containing muscle and a vascular system. The presence of the cyst causes local irritation, so that a fibrous capsule is developed around it by the organ in which it is situated. The cyst contains a clear non-albuminous fluid. Whereas the ovum of *T. solium* or *T. saginata* produces but one larval tapeworm, the ovum of *T. echinococcus* develops into a hydatid cyst, which gives rise to enormous numbers of larvæ. Small white vesicles, called 'brood-capsules,' appear on the inner wall of the cyst. Heads develop inside these capsules as projections outwards of the wall of the brood-capsule. The head is hollow, and its interior communicates with the cavity of the brood-capsule, into which, moreover, the head can be inverted. The endocyst may produce heads directly, as well as by the medium of brood-capsules. One brood-capsule develops several heads. Some cysts remain barren, producing neither brood-capsules nor heads. Each head possesses a rostellum, hooklets, and four suckers, and is identical with the head of a mature worm.

The internal wall of the original cyst may give rise to secondary (daughter) cysts, and the latter may produce tertiary (grand-daughter) cysts. Brood-capsules develop from all these cysts. In that form of hydatids which is most commonly met with in man, the daughter cysts are inside the mother cyst (*endogenous* hydatids); but frequently in animals, and occasionally in man, the daughter cysts bud off outwards and become independent of the mother cyst (*exogenous* hydatids). In a third form, which has been met with in the liver of man, the parasite develops into a mass of little vesicles the size of a pea or less, embedded in gelatinous-looking tissue through the blending of their fibrous tissue capsules (*alveolar* or *multilocular* hydatids). The larger hydatid growths, containing daughter cysts, may attain a weight of from 10 to 30 pounds.

After living in the body for many years it may be, or in

consequence of the fluid being drawn off from the cysts, the parasite dies. It shrinks and becomes fatty and infiltrated with lime salts, so that a mortar-like mass results, in which the hooklets and pieces of the laminated ectocyst may long remain recognisable. Sometimes, however, the cysts rupture into cavities or canals, or on the surface. In other cases, they suppurate, either spontaneously or after rupture.

Hydatids may attack almost any part of the human body, but are most common in the viscera. In more than half the cases the liver is the seat of lesion. The disease is particularly common in Iceland and Australia, where men and dogs are specially close companions.

Symptoms.—Symptoms are often entirely absent. When they are present, they depend largely upon the mechanical pressure exerted by the cyst, so that much depends upon the situation of the latter. Thus a large cyst in the liver may cause no symptoms, whilst a small one in the medulla oblongata may cause death.

Hydatids in the LIVER, if of large size, will cause enlargement of the viscus in a direction corresponding to the situation of the cyst. Thus if the latter bulges upwards, it may give rise to dulness at the base of the right lung. Cysts which are situated in front and superficially sometimes yield fluctuation, and also the 'hydatid thrill' or 'hydatid fremitus'—a sign, however, which is not pathognomonic. This phenomenon is recognised by placing one hand over the cyst, and percussing with the fingers of the other, whereupon a vibration is induced which continues for a little time. Under ordinary circumstances, hydatids cause little or no pain, and the general health remains good.

Rupture occasionally takes place, and if this is into the pericardium, hepatic vein or inferior vena cava, the result is likely to be fatal. Rupture into other parts (*e.g.*, lung or pleura) is sometimes survived. Rupture (and also aspiration) of the cyst may be followed by urticaria, doubtless from absorption of some toxin in the contents.

Suppuration of the cyst constitutes an abscess of the liver, and causes pain, tenderness, fever and emaciation. Here

again rupture may take place, *e.g.*, externally, or into the pleura, pericardium, digestive tract or bloodvessels.

Diagnosis.—An irregular, chronic and painless enlargement of the liver, without impairment of the general health, should suggest hydatids. Fluctuation and thrill are strongly corroborative evidence.

Hydronephrosis is situated lower down; the tumour scarcely moves with inspiration, and is crossed by the colon. Examination of the fluid withdrawn by an exploratory puncture may reveal hooklets in the case of hydatids.

An *enlarged gall-bladder* is less fixed than a hydatid cyst, and the history may throw light on the case.

The irregular enlargement of a *syphilitic liver* is firm, and there may be a history of infection.

If there are signs suggestive of *pleural effusion*, a puncture should be made, and the fluid obtained should be examined for hooklets.

In the LUNG the cyst may exist for some time, and become as large as an orange, without causing either symptoms or physical signs. It may die in the latent stage, but usually, by its continued growth, it ultimately causes symptoms and signs. Cough, hæmoptysis and dyspnœa are the common early symptoms. There may be discomfort about the affected lung, but pain is seldom present unless the pleura is involved. The general health is good. The physical signs include general or local enlargement of the chest, impairment of the respiratory movements, loss of vocal fremitus, the presence of hydatid fremitus (but only in a few cases), and marked dulness on percussion with loss of respiratory murmur over the affected portion of lung. The heart and abdominal viscera are seldom much displaced unless pleural effusion is present.

The cyst generally bursts into the air passages, occasionally into the pleura or elsewhere. The sac may thereupon undergo suppuration. Occasionally the cyst does not burst, but grows until it causes death by its size or by complications. In rare instances it dies spontaneously without rupturing. The dangers of leaving it alone and of operating on it are proportional to its size. The proper treatment

therefore is removal of the cyst and its contents by operation as early as possible.

Diagnosis.—The phenomena just described are not pathognomonic, and exploration may be necessary. But in a country where hydatid disease is common in man, the presence of most of these symptoms and signs, the absence of tubercle bacilli from the sputum, the absence of fever and other general symptoms, the absence of a personal or family tendency to tuberculosis, and the presence of the lesion elsewhere than in the apex, render the diagnosis pretty certain.

Various **complications** may be met with in association with pulmonary hydatids. Pneumonia is common, and may precede or follow rupture. There may be distinctive physical signs, with facial herpes and rusty sputum. Pleurisy, with or without effusion, is less common. Bronchitis is rare. Pneumothorax may result from rupture of the cyst into the pleural cavity, and secondary infection may then lead to pyopneumothorax.

If the cyst actually bulges through an intercostal space, hydatids of the PLEURA may be suspected. If no fluid escapes into the air-passages when the bulging cyst is punctured, the hydatids are probably not in the lung itself. Pleural cysts are usually associated with a pulmonary cyst, less commonly with a hepatic cyst ; but in some instances they are isolated.

Hydatid disease is rare in the KIDNEY. It generally develops in the substance of the organ, but grows till it reaches the pelvis of the kidney. It sometimes ruptures into the pelvis, thus permitting hooklets and daughter cysts to escape down the ureter. Occasionally it grows upwards and ruptures into the lung, or it may burst into the intestine. Sometimes the cyst becomes stationary, and sometimes it suppurates.

The cyst, if large enough, can be recognised as a tumour in the renal region, with the colon lying in front of it. It may grow to a large size without rupturing, but when it bursts, hooklets and daughter cysts appear in the urine, a condition which may supervene in paroxysms during many years. The transit of these materials along the ureter may

give rise to renal colic. If the cyst suppurates, it constitutes an abscess, and this is naturally associated with pain, tenderness and fever.

Diagnosis.—The existence of a renal tumour, a history of renal colic, and the presence of hooklets or cysts in the urine point very strongly to hydatid disease of the kidney.

In *hydronephrosis* the sac will sometimes be found to empty itself for a time, and hooklets are not likely to be present in the urine. Information may in some cases be obtained by puncturing with a hypodermic needle, and examining the fluid thus obtained.

Prognosis.—This is favourable, though the evacuation of the cyst and its contents may not be completed for a long time.

Hydatid cysts occasionally develop in the BRAIN. When symptoms are produced, these point to intracranial tumour.

Treatment of Hydatids in General.—This is purely surgical. Removal of the fluid by the aspirator is frequently sufficient, but the operation must be carried out very slowly, lest daughter cysts be drawn into the cannula and obstruct it. The best practice, however, is to excise the whole sac, with or without previous evacuation, and then, if need be, to drain the resulting cavity. The strictest asepsis must be secured.

4. NEMATODA (ROUNDWORMS).

ASCARIS LUMBRICOIDES (COMMON ROUNDWORM).

This parasite lives chiefly in the small intestine, but may wander into the large bowel. It occasionally passes into the stomach, and may thence reach the mouth, nose, sinuses of the head, Eustachian tube, larynx or bronchial tubes. It may enter the bile-ducts, and may even perforate through the wall of the bowel into the peritoneum. There are usually only one or two worms present, but sometimes they are present in large numbers. They occur chiefly in children.

The parasite is not unlike an earthworm, and is about 10 inches long, the male being smaller than the female. The worm is pointed at each end, and is marked by four longitudinal lines. The male has two chitinous spines at the

posterior end which correspond to the genital orifice ; whereas the genital pore in the female is much nearer the middle of the body. The ova are very numerous and resistant. After fertilisation in the uterus, they pass out in the fæces of the host. There is no intermediate host. The eggs or the embryos reach the intestine of the new host in contaminated drinking water. Children may reinfect themselves through scratching the anus.

Symptoms.—These may be entirely absent. Those that are observed are vague, and include picking of the nose, itching at the anus, grinding of the teeth, emaciation, and even convulsions. Rarer consequences result from the worms wandering for some distance from the small intestine. Thus there may be vomiting of the worm, asphyxia, pulmonary gangrene, jaundice or peritonitis. A bunch of worms may cause intestinal obstruction. It has been supposed by some that symptoms suggestive of enteric fever or of meningitis may be due to roundworms. Ascarides are attracted by foreign bodies, and are sometimes strangled by hooks, buttons and other articles which have been swallowed.

Treatment.—The best remedy is santonin, of which 2 grains may be given to a child, and from 3 to 5 grains to an adult. The powder may be given with milk, sugar, or bread-and-butter, and should be followed by a purge. A better way is to give it in castor oil or syrup of senna ; if necessary, a second dose of the laxative may be given later on. The santonin may be given once a day for three days. It should be remembered that santonin colours the urine yellow; and that yellow vision (xanthopsia), and, after large doses, severe nervous symptoms, may also result.

Oxyuris Vermicularis (Threadworm, Seatworm).

This worm lives chiefly in the large intestine, but may be found in the small intestine, and may even wander to the vagina and urethra. It is most common in children. The female is about two-fifths of an inch long, and has a pointed tail. The male is considerably smaller, and has a blunt tail. It has been supposed that these parasites breed in the

vermiform appendix, as immature forms are often found there. Oxyurides may be present in large numbers, and are readily detected in the fæces after a purge. There is no intermediate host, but the ova do not develop into worms until they have passed through the stomach of the same or of another host. Infection may take place by drinking-water or by vegetables, but is doubtless often a result of scratching the anus.

Symptoms.—These include heat and itching about the anus, especially at night, irritation at the nose, and sometimes restlessness and even convulsions. Females may suffer great discomfort if the parasites reach the vagina.

Treatment.—A strong solution of sodium chloride in cold water should be injected into the rectum every second night for a week or two. Infusion of quassia may be used in the same way. In troublesome cases, santonin and purgatives may be employed to destroy the worms higher up. White precipitate or some other mercurial ointment should be applied to the anus, and if need be to the vulva, to allay itching, and prevent migration of the parasites.

Ankylostomum Duodenale (Sclerostoma Duodenale, Dochmius, Strongylus vel Uncinaria Duodenalis).

The ankylostomum duodenale is a small worm which inhabits chiefly the jejunum or duodenum. The female may be half an inch or more in length, and the male about half that size. The mouth is provided with teeth, by which the worm attaches itself to the mucous membrane. The sexes are distinct, and there is no intermediate host. The parasite is found in Italy, Egypt, Brazil and other hot countries. Epidemic anæmia was met with in French miners more than a hundred years ago, but it was in connection with a deadly outbreak among the workers at the St. Gothard tunnel in 1880 that the disease was first shown[1] to be due to the ankylostomum duodenale. Important outbreaks have been observed among brickmakers in Austria, and in recent years cases have occurred among miners in

[1] By Perroncito of Turin.

Britain. The worm has been termed the 'tunnel-worm'; while *ankylostomiasis*, the clinical condition which it induces, is variously known as 'tunnel,' 'mountain,' 'miner's' or 'brickmaker's anæmia,' 'miner's cachexia,' and 'Egyptian' or 'tropical chlorosis.' The ova are discharged with the fæces, develop in water or damp earth, and after reaching the alimentary canal of the new host in contaminated drinking water, or through the agency of soiled hands, or through the deliberate eating of earth by 'geophagi,' attain maturity in the course of some weeks. There is reason to believe that the embryo worms also reach the intestine by penetrating the skin, travelling by the circulation to the lungs, where they escape from the bloodvessels, then up the air-passages to the pharynx, and finally down the gullet and through the stomach to the bowel.

Symptoms.—The parasites do not necessarily cause symptoms, but if a large number are present, they may collectively suck a great quantity of blood, so that serious anæmia results. Pallor, dyspnœa, palpitation, weakness, œdema, diarrhœa and colic are the principal phenomena. Yet it is known that the symptoms are not always in proportion to the degree of infection, since some individuals may be infected with parasites, and yet suffer little or no inconvenience. The presence of these worms in the intestine is commonly associated with a great increase of eosinophile cells in the blood.

Diagnosis.—The most important points are the geographical distribution and the endemic or epidemic incidence of the anæmia, the occupation of the patient, the eosinophilia and the presence of the ova in the fæces. The stools should be examined microscopically after the administration of thymol by mixing a small quantity of fæces with weak salt solution (1 per cent.).

Prognosis.—This is favourable if the nature of the anæmia is recognised, so that the proper treatment can be put in force. Left to itself, the anæmia usually runs a chronic course, but it occasionally causes death within a few weeks.

Treatment.—The patient is kept on liquid diet for a day,

takes a purge at night, and next morning takes three or more doses (10-30 grains each) of thymol at intervals of an hour. In the afternoon, another purge is taken. To avoid symptoms of thymol poisoning, the patient should rest in bed and abstain from alcohol till the drug has all been evacuated. If thymol fails to destroy the parasites, male-fern should be tried. The anæmia may subsequently call for treatment by iron.

Trichina Spiralis.

Trichina spiralis occurs in its mature form in the intestine of man and many other animals, and in its immature form in the muscles. Man is infected by eating imperfectly cooked trichinosed pork, and swine are usually infected by eating trichinosed rats.

The worms are cylindrical in shape, and the anterior end is more pointed than the posterior. The mature female is one-eighth of an inch long. The male is smaller and is furnished with certain projections at the tail, close to the orifice of the genital duct. When infected pork is eaten by man, the capsules containing the embryos are dissolved in the stomach, and the worms reach the intestine. In two or three days they become mature and pair. The ova develop into embryos in the uterus, so that reproduction is viviparous. The embryos begin to be born a week after infection, and in the next few weeks, each female may give birth to a thousand or more. The adults survive for a few weeks only. The embryos travel to the voluntary muscles by way of the intestinal wall, the subperitoneal tissues, and possibly in part by the blood-stream. They take up their position within the sarcolemma or sheath of the individual muscle fibre, and in about a month after the infection they have become encysted. The cyst is due to interstitial myositis set up by the irritation of the parasite. Two or three embryos are occasionally present in one cyst. After a time the cyst wall becomes calcified, and if the worm should die, the contents of the cyst may also become infiltrated with lime salts. The embryo may live for many years in the muscle, but it undergoes no further development

until the muscle is eaten by the new host. To the naked eye, the affected muscle looks as if dusted with white specks. In examining swine's flesh for trichinæ, the muscles which should be specially observed are those of the tongue and larynx, the diaphragm, and the intercostal and abdominal muscles; but those of the limbs may also be infected. Thorough cooking of the flesh kills the trichinæ.

Symptoms (*Trichinosis* or *Trichiniasis*).—The severity of the symptoms depends upon the number of trichinæ. With only a few worms reaching the muscles, no symptoms may be induced. In well-marked cases two stages are recognisable. (1) In many cases, gastro-intestinal catarrh sets in some days after infection and continues for about a week. It is characterised by anorexia, abdominal pains, vomiting and diarrhœa. (2) As the first stage subsides, pains in the muscles and considerable fever develop; these may easily be mistaken for rheumatism. The muscles are swollen, hard, tender and partly paralysed. Œdema appears in the eyelids, and spreads over the face to the limbs. The involvement of the muscles may interfere with mastication, deglutition, phonation and respiration. The general course of the temperature may resemble that of enteric fever, and in severe cases a considerable degree of anæmia and emaciation may develop. Leucocytosis is present, and there is marked eosinophilia. After four or five weeks the symptoms gradually subside, and after all the worms are encysted the patient recovers completely. But sometimes the attack ends fatally from diarrhœa, peritonitis, pneumonia, asphyxia (from embarrassment of the diaphragm and intercostal muscles) or exhaustion.

Diagnosis.—This can generally be made from the sequence of intestinal and muscular symptoms. Œdema of the face and eosinophilia constitute additional evidence of great value. Moreover, adult worms may be found in the stools; embryos may be found in portions of the suspected food; and if there is still doubt, a portion of an affected muscle may be harpooned (under local anæsthesia) and examined.

Treatment.—If it is known within a few days that trichinosed food has been taken into the stomach, emetics and

purgatives should be administered until the contents of the alimentary canal have been thoroughly evacuated. Even after the muscular pains have begun, if there is not diarrhœa, calomel and salines may be given with advantage, as experience shows that early diarrhœa is a favourable feature. There is no specific which is known to destroy the worms without damage to the patient, but male-fern, santonin or thymol may be tried in the early stages. It is important to maintain the general strength by abundance of light food, and if need be, by stimulants.

Trichocephalus Dispar (Whipworm).—This worm is very common in France, but rare in England. The anterior part is thread-like; the posterior part is much thicker. The posterior part is coiled in the male, straight in the female. The whole worm measures an inch or more in length. It lives chiefly in the cæcum. There is no intermediate host, but the ova pass out with the fæces, and into the stomach of the new host. No symptoms are present, as a rule, but intestinal catarrh and diarrhœa may be induced. If treatment is necessary, santonin is the remedy.

Strongylus Gigas (*Eustrongylus gigas*) occurs in dogs and other animals. In a few instances it has been found in the pelvis of the human kidney. The female measures a yard, and the male a foot in length.

Strongylus Bronchialis (*Filaria bronchialis*), which may cause serious disease in the lungs and bronchi of certain lower animals, has occasionally been found in the air-passages in man.

Strongyloides Intestinalis (*Anguillula intestinalis*, *A. stercoralis*, *Rhabdonema intestinale*) is found in the stools in the diarrhœa of hot countries. When present in great numbers in the intestine, this parasite may be the cause of diarrhœa.

Filaria Sanguinis Hominis.

Filaria sanguinis hominis is the *larval* or *embryonic stage* of a parasite of which at least four species are recognised.

To study the infected blood, Manson recommends that half a drop be spread on a slide, over 1 square inch, and allowed to dry. It may then be stained at once, or after any length of time, by immersion for an hour in a weak solution of eosin, fuchsin or any other aniline dye (1 drop of the saturated alcoholic solution in 1 ounce of water), after which it is washed and mounted. In this way the filariæ and leucocytes are stained. To display the structure of the parasites, the film should be dried, fixed in absolute alcohol, and stained in weak hæmatoxylin. The preparation should be examined with a very low power in the first place. The embryos as seen in fresh blood are slender, snake-like creatures, which wriggle with great activity, and may be kept alive for several days if drying is prevented. They manifest no definite internal structure, but have a dark granular appearance, and are quite loosely invested by a structureless sheath. They measure $\frac{1}{80}$ by $\frac{1}{3500}$ of an inch.

(1) *Filaria nocturna* is present in the blood of superficial parts at night only. In the daytime its retreats are the lungs, the large arteries, and sometimes the capillaries of the heart; but not the bone-marrow, spleen or liver. If the infected person changes his habits so as to rest by day and be awake at night, the parasites change their habits in the same way. This periodicity is supposed to be an adaptation to the nocturnal habits of certain kinds of mosquito (*Culex* and *Anopheles*) which swallow the embryos along with the human blood, and act as *intermediate hosts*. In the insect's stomach, the sheath is thrown off, and the parasite then penetrates to the thoracic muscles of the mosquito, where it grows in size and acquires an alimentary canal. It then travels forward to the proboscis of the insect, and thus, it is highly probable, reaches its human *definitive host* when the mosquito bites; though it is conceivable that infection may take place by way of the stomach, through the insect being drowned and liberating the parasites into drinking water. Having reached the body of man, they penetrate into the tissues, and especially the lymphatics, where they are found in their *mature phase*, known as *Filaria bancrofti*.

This parasite is found in many warm climates, and the

adults, which live together in the lymphatics, may survive for many years. The females are about 3 inches long, the males about half that length. They look like white horse-hairs. Reproduction is viviparous, and the embryos having escaped into the lymph stream, pass through the lymph glands and on into the blood. Various pathological conditions to be subsequently mentioned are related to the presence of this parasite in the body.

(2) *Filaria diurna* is present in the peripheral circulation in the daytime only, probably in correspondence with the habits of its intermediate host. The parasite is met with in tropical Africa, and is supposed to be the embryonic form of *F. loa*. The latter moves about under the skin, and may cause some inflammation.

(3) *Filaria perstans* is found in the superficial vessels both by day and by night. It occurs on the West Coast of Africa and in British Guiana. The mature forms have been found in the subperitoneal and subpericardial connective tissues. The parasite is often found in individuals who show no symptoms.

(4) *Filaria demarquayi* (possibly identical with *F. ozzardi*) occurs in the West Indies and New Guinea. Like the last form, it is found in the circulation both by day and by night. The adults have been found in the subperitoneal connective tissue. No pathological results are known.

Filariasis or filarial infection is in a large proportion of cases unassociated with symptoms. Symptoms result, as Manson has shown, from some departure from the normal in the life-history of the parasite, and especially from abortion on the part of the adult female, whereby ova instead of active embryos are liberated into the lymph stream. The ova are much thicker than the embryos, and are apt to block the lymph channels at the glands. Or, again, the parent worm may itself obstruct the thoracic duct, or may give rise to inflammatory stenosis of that duct.

In *chyluria*, the urine suddenly becomes milky in appearance, and often gives off a whey-like odour. Occasionally the urine is reddish, probably from the admixture of blood (*hæmatochyluria*). After standing for a short time, it

coagulates into a jelly, and it readily decomposes. Clotting may take place earlier, and micturition may be temporarily arrested by blocking of the urethra. Albumen, fat and fibrin are present in the urine. *Filaria nocturna* is generally to be found in the blood at night, and in the urine at any part of the twenty-four hours. The attack may be accompanied by aching in the loins and by feverishness, as well as by interruption of micturition.

Chyluria is believed to be due to rupture of lymphatics in the urinary tract, after these vessels have been obstructed and rendered varicose through the agency of adult filariæ, or their ova. The condition comes and goes in the most irregular and unaccountable manner, and cure can scarcely be expected.[1] Drugs cannot be relied upon in **treatment.** Manson recommends rest in the recumbent posture with the pelvis elevated, restriction of fluids, and complete avoidance of fat in the food.

Abscess may result, either superficially or in the deeper tissues, in consequence of the irritation set up by a dead adult worm (*F. bancrofti*), or after blocking of lymphatics by ova.

Varicose lymphatic glands are due to filarial obstruction. The femoral and inguinal glands usually suffer on both sides, and constitute soft painless swellings over which the skin can be freely moved. The swelling is less in the recumbent than in the upright posture. The **diagnosis** may be made by examination of the blood, and, if need be, by exploratory puncture of the swelling. In ordinary circumstances, no special **treatment** of these glands is necessary, or at most an elastic bandage may be applied ; but if they become painful or inflamed, they should be excised.

Lymph scrotum is characterised by a varicose condition of the lymphatics on the surface of the scrotum. The dilated vessels may be scanty or very numerous. Sometimes they rupture, either spontaneously or through injury, and allow great quantities of chyle or lymph to escape. Inflammation, abscess and high fever are common complications, and elephantiasis may ultimately supervene. In the simple cases

[1] It is to be noted that chyluria is not always due to the filaria.

no treatment is necessary beyond protection and cleanliness, but in troublesome cases the diseased tissues should be excised.

Chylous hydrocele may occur with or without lymph scrotum. The treatment is by simple tapping.

Elephantiasis (*Elephantiasis arabum*) is endemic where *Filaria nocturna* occurs, and is believed to be due to that parasite, although in most cases it is no longer to be detected in the peripheral circulation. Elephantiasis is by far the most common manifestation of filariasis. It is most common in adult life, and in the dark races. The sporadic form, which occurs in any part of the world, is not necessarily due to the filaria, though brought about by lymphatic obstruction.

Morbid Anatomy.—In most cases there is simply a hypertrophy of the normal tissues, including the bloodvessels and lymphatics ; but in other instances the lymphatic structures, including the vessels, spaces and glands, are specially involved, so that a soft swelling is produced which, on being punctured, yields chyle or lymph (*nævoid elephantiasis*). The local changes are most commonly observed in the lower limbs and genitals. The skin becomes thickened from overgrowth of its fibrous elements, and is thrown into folds and lobules. Underneath the skin there is great development of a loose, juicy connective tissue, and this overgrowth may even extend as deeply as the periosteum and lead to thickening of the bones. The toes and penis may be buried in the great tumours thus formed. The scrotum may weigh over 100 pounds.

Symptoms.—There is localised inflammation of the skin and deeper soft tissues in the affected region, characterised, as usual, by redness, swelling, pain and elevation of temperature. The skin may yield a serous discharge, and dilated lymphatic vessels may rupture and give exit to lymph. When the scrotum is affected, there may be severe pain in the testes, spermatic cords and loins ; and acute hydroceles may develop. The general symptoms include high fever, rigors, vomiting and delirium, which may pass off after hours or days with profuse perspiration (*elephantoid fever*). The tendency is for these febrile paroxysms or *erysipelatoid*

attacks to recur every few weeks or so, and on each occasion to leave the local overgrowth greater than before ; but occasionally there is a slow progressive hypertrophy without pain or fever.

Prognosis.—The disease does not shorten life.

Treatment.—In the febrile stage, diaphoretics should be given, together with opiates if the pain is severe. Fomentations may be applied locally. Once the paroxysm is over, the most important thing is removal from the endemic area, a procedure which, in the early stages, may permanently arrest or even cure the disease. Quinine and iron internally, and bandaging of the affected parts may be of some service. Surgical treatment is often very successful, especially in the case of tumours of the genitals ; and complete removal of the hypertrophied tissues may rid the patient, not only of the local lesion, but also of the recurring febrile paroxysms.

The use of the mosquito net is essential in filarial just as in malarial countries.

Filaria Medinensis (Dracunculus Medinensis, Guinea Worm).

The Guinea worm is met with in Guinea, Abyssinia, India and other countries. The female only is known at present. The mature worm is 2 or 3 feet long, and about a twelfth of an inch in diameter. It lives in the subcutaneous tissue, especially of the feet and legs, and produces great numbers of young in a viviparous manner. The symptoms produced by the worm (*dracontiasis*) are practically confined to this part of its life-history. In order that the embryos may develop further, they must reach water ; and this fact no doubt explains the selection of the lower limbs, which in natives are most likely to be wetted. The presence of the worm leads to the development of a pustule which ruptures, and through the ulcer thus produced, the parasite discharges the embryos. This process is completed within a few weeks, and the worm itself will often leave spontaneously thereafter. Sometimes it dies before parturition takes place, and causes no symptoms.

When the embryos reach water, they bore into the body of *Cyclops*, a minute crustacean, and grow there. With their intermediate host they are next taken into the alimentary canal of man, and it is supposed that here they become sexually mature and copulate, the males dying immediately afterwards. The females, on the other hand, penetrate to the subcutaneous tissue and generally travel, as already explained, to the foot or leg.

Treatment.—The usual treatment is to allow the worm to discharge her embryos without interruption, since serious trouble may follow if the parasite is broken and the embryos escape into the tissues. After parturition is complete, the protruding part of the worm may be rolled round a small piece of wood, so as to maintain slight traction and prevent retraction. Each day a little more of the worm may be wound on the stick. Another method is the injection into the worm as soon as it is recognised, or into several spots around it, of corrosive sublimate solution (1 in 1,000).

5. ARACHNIDA

Acarus Scabiei (Sarcoptes Hominis, Itch Mite).

This acarus is a small animal, somewhat like a tortoise in shape, and just visible to the naked eye. Spines are present on its back. Of the eight legs with which the mature animal is furnished, the four in front are provided with suckers. In the female all the four posterior legs have setæ or bristles, but in the male the two median posterior legs have suckers. The male is smaller than the female. The young do not possess the sexually distinctive posterior median limbs, and therefore have only six legs till they have moulted repeatedly.

The male acarus lives on or near the surface of the skin. The unimpregnated female commonly wanders on the surface. The impregnated female bores into the skin, and in less than half an hour takes up her position in the deep layers of the epidermis. Thereafter she lays from ten to fifteen eggs at the average rate of one each day. Each day she burrows a little further so as to leave room in the burrow

or *cuniculus* for the next egg. She cannot recede owing to the backward projection of her dorsal spines, and she dies in the burrow after laying her eggs. The eggs are hatched in about a fortnight, probably about the time when the part of the burrow which they occupy approaches the free surface of the skin. The larvæ burrow into the skin even more deeply than the impregnated female, and often cause vesiculation.

Symptoms.—*Scabies* or *Itch* is the skin disease excited by *Acarus scabiei*. It is highly contagious, and readily passes from an affected person to another individual occupying the same bed. The hand of a nurse may transmit it to the hips of a child. The parasite attacks by preference skin which is delicate in texture and protected by clothing. Thus the skin between the fingers, the anterior surface of the wrist, the penis, the nipple of the female, the lower abdomen, the thighs, and the buttocks are favourite places ; but any part may be involved except the face and scalp of adults. The eruption is really an artificial eczema, associated with much itching, especially at night, and aggravated by the consequent scratching. The distribution is symmetrical and the lesions are polymorphous (papules, vesicles, pustules).

Diagnosis.—The distribution of the eruption is often very suggestive, involving as it does the lower portions of the upper limbs and trunk, and the upper portions of the lower limbs. The presence of lesions on the penis or nipple is also very significant. A history of infection or of a possibility of infection is frequently obtainable. The burrow or *cuniculus* looks somewhat like an old scratch, and if a pin be inserted, or if the burrow be opened up with a penknife, the parasite may itself be obtained for examination, since it usually grasps the article with which it is touched.

Treatment.—The usual treatment is by sulphur in some form. Before going to bed the patient should take a hot bath to clean the skin thoroughly, and after drying the surface, should rub in about an ounce of sulphur ointment, which should be applied to all parts except the face and scalp. This process of bathing and inunction should be performed on three successive nights, after which the sulphur

should be stopped lest a new artificial eczema be induced. The infected clothing must of course be changed and thoroughly washed.

Sometimes a sulphur bath is used (8 ounces of sulphurated potash in 30 gallons of water); it should be repeated every few days for a fortnight.

McCall Anderson recommends the following as destructive to the acari, soothing to the skin and possessed of a pleasant aroma: R. styracis liquidi, ʒi.; adipis, ʒii. Melt and strain. Or balsam of Peru may be employed in combination with twice its weight of lanolin.

Acarus Folliculorum (*Demodex folliculorum*, *Steatozoon folliculorum*, *pimple mite*) is a small arachnid which is found in the sebaceous glands of the face and other parts. It has eight legs, each of which is armed with three claws. It is apparently not pathogenic.

Leptus Autumnalis (*harvest bug* or *mower's mite*) is a red parasite which occurs in the fields and in gardens, and may attach itself to man and animals by its head. It may cause a good deal of irritation of the skin.

The importance of *ticks* as transmitters of disease has been alluded to in connection with relapsing fever (see p. 41) and piroplasmosis (see p. 216).

6. INSECTA

Pediculus Capitis (Head-louse).

This parasite is met with only on the head, except in rare cases. It crawls about the scalp, and in this way, as well as by attacking the skin, gives rise to itching and scratching. The adult louse is about $\frac{1}{12}$ of an inch long, and has the head, thorax and oval abdomen distinct. The head has two antennæ and prominent black eyes. The thorax is furnished with six legs armed with claws for grasping the hair. The male has a conical penis on its back. The female is larger, and has its posterior end notched. The ova ('nits') are deposited on the hairs, and are hatched in five days. The pediculus does not bite, but inserts its proboscis into a follicle or through the skin, and lives by sucking blood in this way.

Pediculosis (*phthiriasis*) *capitis* is most common in children. The serum exuding from the scratches may be enough to glue the hairs together into crusts, but eczema or impetigo may be added to the original condition, and this may spread to the neck and ears, and be associated with enlargement of the lymph glands. A pustular eruption limited to the back of the head is almost pathognomonic of pediculosis capitis. *Plica polonica* is a condition of firm matting together of hair, lice, scabs, pus, and dirt, such as results from extreme neglect.

Treatment.—This consists in cleaning the head and applying a parasiticide. Whenever practicable, the hair should be cut as short as possible, or the head may be shaved. Failing this, the pediculi may be killed by applying carbolic lotion under a waterproof covering. Thorough combing is necessary to remove the nits, and as a preliminary, the hair may be soaked for some time with dilute acetic acid, or with a solution of borax or sodium carbonate. A weak mercurial ointment, or stavesacre ointment, is a suitable parasiticide.

Pediculus Corporis (P. Vestimenti, Body-louse, Clothes-louse).

This species occurs on the non-hairy parts of the body. In the main it is very similar to the head-louse, but it is considerably larger. It lives in the folds of the clothing, and there it deposits its nits or ova, which are little, yellowish, somewhat crystalline-looking bodies. It specially favours parts such as the shoulders and waist, where the garments are in close contact with the body. It attacks chiefly those who are old, debilitated or uncleanly in their habits.

The insect does not bite, but sucks blood through a membranous tube which it inserts into a sweat pore. The tearing by the patient's nails of the hyperæmic follicles which persist after the insect has fed, gives rise to one of the most characteristic signs of *Pediculosis* (*phthiriasis*) *corporis*, viz., reddish-brown crusts, which are due to exuded blood. The eruption, therefore, is 'pruriginous.' The fact that it is

absent from the hands is enough to distinguish it from *scabies*. The deeply pigmented, harsh and indurated condition of the skin often seen in neglected adults, tramps, and others, and known as *vagabond's disease*, is attributable largely to pediculi, though no doubt due in part to other parasites, as well as to exposure and habitual want of cleanliness.

Treatment.—This consists in personal cleanliness, disinfection of clothing, and the application of stavesacre ointment.

Pediculus Pubis (Phthirius Pubis vel Inguinalis, Crab-louse).

This louse is smaller than either of the other two species. It occurs on all hairy parts except the head, but especially about the pubic region. The body is broader in proportion to its length than in the case of the head-louse, and the demarcation between thorax and abdomen is not distinct. This parasite clings to the root of the hair, and its ova are also attached to the hair close to the skin.

The irritation caused by the insect leads to itching and scratching, and sometimes to a papular eruption. The faint, bluish, non-elevated spots sometimes observed on the trunk and limbs, and known as *taches bleuâtres*, or *maculæ cæruleæ*, have been regarded as pathognomonic of the presence of *Pediculus pubis*.

Treatment consists in the inunction of white precipitate ointment.

Cimex Lectularius (Acanthia Lectularia, Common Bed-bug).

This insect is reddish-brown in colour, and rounded and flattened in form. It has an offensive smell, which is specially noticeable when it is crushed. It lives about bedding, furniture, cracks in the walls, etc., and comes out particularly at night to suck blood. The victim may or may not be conscious of a slight sting, but the wound is soon followed by the development of an urticarious wheal, which is distinguishable from that of ordinary urticaria by the

presence of the bite in the centre. One insect may cause several bites, and the intense itching and consequent scratching may lead to well-marked secondary changes in the skin.

Treatment.—To relieve the itching, vinegar may be applied, or a lotion consisting of 2 drachms each of dilute hydrocyanic acid and glycerin, with 6 ounces of rose-water.

Pulex Irritans (Common Flea).

This brownish-red insect bites by means of a bristle-like tongue and two sword-like maxillæ. It lays its eggs on furniture, clothing, etc. When it attacks the skin, it causes a minute hæmorrhage and transient hyperæmia. Some people are not inconvenienced by such bites, whilst in others urticaria may develop and give rise to intense itching which causes the victim to tear his skin.

In children freshly admitted to hospital, it is sometimes difficult to distinguish between flea-bites and *purpura*. Quite recent bites show a hæmorrhage surrounded by a zone of hyperæmia After the hyperæmia has passed away, the different bites will be found to possess a uniform size. In purpura there is no surrounding hyperæmia, and the spots are not exactly alike. Fleas or their fæces (small brown specks) may be found on the clothing.

Vinegar may be applied to allay the itching due to flea-bites in children.

Pulex Penetrans (Sand-flea, Jigger, Chigoe).

This is a small brownish-red parasite which attacks man and the lower animals in tropical countries. The impregnated female enters the skin about the toes, and may cause a pricking sensation in the process. In the course of a week or ten days—owing, it is supposed, to the great distension of the generative organs of the insect—œdema, pustulation, and occasionally lymphangitis ensue.

Treatment.—The flea should be killed and extracted by a heated needle, after which an antiseptic dressing should be applied.

Culicidæ (Gnat Family).

This family of diptera consists of the gnats, midges and mosquitoes, and is of great importance because mosquitoes are the agents by which the parasites of three diseases, viz., malarial fever, yellow fever, and elephantiasis, are transmitted to man. Male mosquitoes are believed to feed on fruit only, but the females feed also on blood which they suck from men, beasts and birds. The females appear to require a meal of blood before they lay their eggs. The proboscis contains a number of sharp stylets, which are plunged into the skin. Before the female actually sucks up the blood, she injects saliva into the wound through one of the stylets. The saliva causes the irritation of the bite, and also conveys the spores of the malaria parasite.

Of the hundreds of species of culicidæ, three genera are of special importance: *Culex*, which is related to elephantiasis; *Stegomyia*, which is related to yellow fever; and *Anopheles*, which is related to malaria and to elephantiasis.

The great importance of certain other *flies* (particularly species of *Glossina*) as transmitters of disease has been indicated in connection with different varieties of trypanosomiasis (see pp. 213, 214).

Parasitic Dipterous Larvæ.

The larvæ (maggots, bots, or gentles) of certain dipterous insects are occasionally found in man, the condition being then known as *myiasis*. Thus the larvæ of the bluebottle or of some other fly may be found infesting wounds, the nose, ears, etc., in neglected and debilitated subjects. Such a wound is said to be 'living' (*myiasis vulnerum*, *narium*, etc.).

In hot countries, the bot-fly of man (*Œstrus hominis*), and the bot-flies of cattle, sheep, and deer give rise to *cutaneous myiasis* in man. Subcutaneous abscesses are produced.

Another form of the infection is *gastro-intestinal myiasis*, the larvæ being vomited, or evacuated per anum. Cases in which larvæ belonging to one or more species of *anthomyia* have been passed by the bowel have been recorded by Fin-

layson and R. M. Buchanan of Glasgow[1]; but various other genera may be represented. The infection in these instances may take place through the medium of stale, uncooked vegetables, raw meat, drinking-water, etc., or possibly from the fly depositing its eggs on the mucous membrane of the anus at the time of defæcation.

The **symptoms** which may be present in different cases include vomiting, diarrhœa, abdominal pain, anorexia and epileptiform phenomena. In rare cases the disease is chronic, and associated with inflammatory changes in the bowel, and with severe constitutional disturbance.

Treatment.—When the cause of the symptoms is recognised, the treatment indicated would include an emetic, purgatives, bitter tonics by the mouth, and enemata of salt solution.

[1] *Trans. Glasg. Path. and Clin. Soc.*, February, 1898

APPENDIX

Mediterranean Fever (see p. 168).

While this work was passing through the press (March, 1906), the Royal Society published a further report of the Commission appointed by the Admiralty, the War Office, and the Civil Government of Malta, to investigate Mediterranean fever. In a previous report it was shown that Maltese goats are susceptible to the disease, since the *Micrococcus melitensis* occurs in their milk. The Maltese obtain most of their milk from goats, and they use it uncooked, so that the infection of man by way of the alimentary canal through the use of goat's milk is easily intelligible. Indeed, it is now regarded as certain that this mode of infection is largely responsible for the occurrence of the disease among the population.

It has further been determined that certain species of mosquito in Malta act as carriers of the microbe, and it is highly probable that these insects become infected by biting infected human beings.

Among the preventive measures which are recommended is the oiling of stagnant waters to destroy the larvæ of mosquitoes.

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